CERTIFICATE

This is to certify that this dissertation work on **"UKKARA SOOLAI"** has been carried out by **Dr.K.SAMRAJ** during the year 2010-2013 in the **Post Graduate Department of Maruthuvam, Government Siddha Medical College, Chennai-600106** under my guidance and supervision in partial fulfillment of regulation laid by **The Tamilnadu Dr. M.G.R Medical University, Chennai** for the *final M.D* **(Siddha) Branch I- MARUTHUVAM** examination to be held in **April 2013**.

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A STUDY ON

UKKARA SOOLAI

the dissertation Submitted by

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under the Guidance of

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Contents

INDEX

CONTENTS

Introduction

INTRODUCTION

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«¾¢Ã ÅÕÅ§¾¡÷ §¿¡ö
-¾¢ÕÅû Ù Å÷.

In South India, the Siddha system flourished earlier than the other systems. And also it is one of the oldest systems in India. The term ‘Siddha’ means Achievement and the Siddhar were saintly figures who have contributed to the development of this medical system.

Siddhar is a Tamil word that is derived from its root Chiti. This means Perfection in life Or Heavenly bliss. It generally refers to eight kinds of paranormal powers (Ashtama chittigal) attainable by man, that persons are known as Siddhar. Every prominent temple in Tamil Nadu is attributed to those great Siddhars.

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Á ñ Ï ÖÕÅ¡ ¸¢ Å¡ÛÄÅô- À ñ Ï Å¢¾
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Siddhars knowledge in Iatro-chemistry, minerals, metals and plants were awesome. The process like oxidation of mercury, minerals, metals and the preparation of herbal medicines which cure the diseases and rejuvenate the entire human system.

According to Siddha science to prevent the ageing process, they discovered and used various kinds of medicine faculties like Muppu, Kaya kalpam, Yoga and also Internal medicines.

“§ÀÚ þÇ ¨ Á þ ý Àõ À½ ãôÒ º¡ ì ¸¡ Î ¬ Ú í ¸ÕÅÄ ¨ ÁôÒ”

Preventing the ageing means to prevent the body from the various degenerative diseases occurring in old age. Siddha medicine places a major role in maintaining health of the elderly people. This medicine has potential to emerge a mainstream in health care delivery system of the geriatric community.

Geriatric is a branch of medicine which emerges in the last two decades as a separate entity towards the health care of the elderly is called as ‘Moopu Iyal’ in Siddha medicine.

The term geriatric derived from Greek. ‘Geron’ means old man, ‘Iatrica’ means the treatment of disease. The problems of geriatrics include altered endocrine functions, decreased functions of nervous system, tissues, and organs. Those problems lead the processing of ageing.

In elderly males, they got so many degenerative problems. The major encountered problem is difficulty in voiding urine. Usually men above 60 years are affected due to the enlargement of prostate gland.

The enlargement of the central part of the prostate narrows the calibra of the urethra passing through its middle and obstruction occurs that leads to stagnant micturation. Sometimes the enlargement predict as a malignant tumor. This is literally called as benign prostatic hyperplasia (BPH).

The incidence of BPH increases according to the age. It is estimated that 65% of men at the age of 60 were suffering from BPH. The prevalence of disease is 8% in the age of 31 – 40 years, 40 – 50% in 50 – 60 years and 80% in above 80 years. The symptoms includes, delayed emerging of urine, dysuria, increased frequency of urine, urgency, urinary incontinence, nocturia and drippling of urine and lower abdominal pain at advanced stage.

The complications of the diseases are very threatful to life. Those are recurrent urinary infection, which leads to cystitis and rarely kidney failure. Severe pain in lower abdominal area, rectal obstruction, Prostitis, Urithritis are common.

In Siddha disease classification of this symptoms are correlated to “UKKARA SOOLAI” which is one of the 15 types of Soolai Noi.

In modern treatment UKKARA SOOLAI (BPH) is curable by surgery only. I prefer to select this topic for my dissertation to reduce the symptoms by a metallic Sasthric preparation of “VELVANGA PARPAM”.

Aim & Objectives

AIM AND OBJECTIVES

AIM:

The aim of the study is to conduct a clinical trial on **Ukkara Soolai (Benign Prostate Hyperplasia)** patients with a combination of siddha drug **Velvanga Parpam** and to improve the happiness of geriatric people.

OBJECTIVES OF THE STUDY:

- To review, the siddha literary evidences dealing with etiology, classification signs and symptoms, diagnosis, diets, prognosis of Ukkara Soolai in siddha system of medicine.

- To identify the clinical symptoms which resemble the modern clinical presentation with description of Ukkara Soolai is related with benign prostate hyperplasia.

- To gather an idea on the incidence of Ukkara Soolai with regard to age, severity of signs and symptoms, diets, habits and other old age diseases.

- To access the role of well known siddha drugs to control all the symptoms and sign of Ukkara Soolai.

- To systematically record the sign and symptom and the test results of full evaluation.

- To assess the role of diagnosis tools of siddha system in the follow up as prognosis in clinical Ukkara Soolai.

- To study the
    - Acute and sub acute toxicity
    - Pharmacological activity of the Siddha formulation.
    - And Bio-Chemical analysis.

- To conduct the clinical trial on the patients of Ukkara Soolai with a trial drug of ***Velvanga Parpam.***

- To find out the statistical analysis of the clinical study.

- To handle the modern parameters to confirm the diagnosis and prognosis of the study.

Review of Literature

Siddha Aspects

REVIEW OF LITERATURE

SIDDHA ASPECT

Siddha, the oldest system of Indian medicine has gone deep into the problem of the elderly people and it proposes mainly the ‘Kayakalpa’ treatment. The term ‘Kayakalpa’ is derived from two Tamil words ‘Kaya’ which means the body and ‘Kalpa’ means the stone. Keeping the body as strong as a stone is the main aim of Kayakalpa. By this treatment the body gets free from the graying of hair, ageing, diseases etc.

In siddha, kaya kalpa treatment helps to prevent the ageing. But now a days ageing process in human being is a regular one. It includes degenerative diseases, hormonal diseases, neuro-muscular diseases, metabolic diseases etc. These ageing processes is called “MOOPPU” in siddha system and “GERIATRIC” in western medicine. The term *geriatrics* comes from the Greek, *Geron* meaning "old man" and *Iatros* meaning "healer". However, geriatrics is sometimes called medical gerontology.

Life style modification is also an integral part of geriatric and “kaya kalpa” treatment. It includes proper food habits, sleep and relaxation, exercise, personal cleanliness, tension free and clear mind, entertainment etc. ‘Yoga’ and ‘Pranayama’ have got their own significance in dealing with this challenge. Research have proved, beyond doubt that these are efficacious in neuro-muscular, musclo-skeletal, respiratory, and psychosomatic and other health problems in elderly people.

UKKARA SOOLAI

SOOLAI:

DEFINITION:

In general, *Soolai* can be defined as pricking or piercing pain that may occur in any part of the body. It is named according to the part of the body it arises. **Ukkara soolai** is defined as pain arising during the time of Micturation or when the bladder is filling only in males.

ETIOLOGY:

Etiology for *Soolai* has been described by many siddhars. They are as follows,

General etiology for *Soolai* according to

a) Yugimuni in Yugi vaithiya Sinthamani800:

"சார்வான சூலைவரு மாறுகேளாய்
தக்கசிறைப் பட்டிருக்குந் தீமை யாலும்
ஆர்வான வறச்சுடுசோ றருந்த லாலும்
அறவுமே சலிப்பாலு மோட லாலுந்
தார்வான சபைமிகுந்த சண்டையாலும்
தகையான துவர்ப்பொசிப்பு புகைத்த லாலும்
வேர்வான மோகத்தின் புணர்ச்சியாலும்
மிகுந்தபசி யுறுதலினாற் சூலை யாமே"

--யூகி வைத்திய சிந்தாமணி 800-பக்கம் 84

"ஆமென்ற வன்னத்துக் கிறுதி பண்ணி
யதிகபர தேசிகளை யடித்த பேர்க்குங்
காமென்ற கற்புடைய மங்கை மாரைக்
கருதியே மனத்துளிச் சித்த பேர்க்கும்
வாமென்ற வாழ்மரத்தை வெட்டி னோர்க்கும்
வழிமறித்து பொருள்பறித்த மதிகே டர்க்கும்
ஏமென்ற எச்சிறனைக் கவர்ந்த பேர்க்கும்
இகத்திலே நோவெய்திச் சூலை யாமே"

--யூகி வைத்திய சிந்தாமணி 800- பக்கம் 85

- Imprisonment
- Consumption of hot diets
- **Depression**
- Exhaustive running
- Anxiety
- Excessive consumption of astringent diets
- **Indulgence in excessive intercourse**
- Excessive starvation
- Ill treating the physically and mentally deserving people
- Deforestation
- Winding other people things (Robbery)
- Giving their eaten left over's to others

b) **Guru naadi sasthiram:**

"சூலை வரும்வாறதனைச் சொல்லக் கேண்மோ
சுற்றியதோர் விசைநரம்பில் வேவுகண்டு
காலையுமே யூடுருவித் தமர்தான் விம்மிக்
களிப்பதுபோல் மாசுபற்ற யிரத்தம் வற்றி
மாலையது போல்தொடுத்து நரம்பின் மீதே
வகையான விசைநரம்பை மடக்கிக் கொள்ளும்
கோலைவிட்ட குருடரைப்போல் திட்டமிந்து
குடிக்கெடுக்குஞ் சூலையது குறிகண்டீரே"

- குருநாடி நூல்- பக்கம்-235

Vatha force ,one among the three vital force is disturbed and penetrates through the nerves and affects the Senneer thathu(Blood) resulting in deterioration of blood and produces stress in the body. The *Soolai* settles in the body as a blind man without stick and destroys the body and life.

c) **Agathiyar in Gunavagada thirattu:**

"திருத்தமாய் வாதத் தோடே
தீங்கொடு பித்தஞ் சேரில்
பொருத்துகள் தோறும் நொந்து
போதவே பிடிக்குஞ் சூலை
பொருந்திடா வாயு கொள்ளும்
பேசிய வாறே நோகும்
கருத்தினால் அறிந்து நன்றாய்
கண்டிடாய் மனிதர்க் கெல்லாம்"

- அகத்தியர் குணவாகடம்- பக்கம்-26

The above literature describes that the dearrangement of Vatham along with Pitham will cause pain in all the joints and this condition is indicated as *Soolai.*

d) **Thanvanthiri in Therayer vagadam:**

"அங்கத்திலெண்ணெய் தேய்த்து முழுகும்நா ளரிவைசேரிற்
சங்கத்திற் சுக்கிலத்தைத் தம்பிக்கிற் றுலைநடந்த
பங்கத்திற் பசியிற்கட்க பதார்த்தத்தில் மிகும்புளிப்பில்
வங்கத்தியாச தோஷத்தில் வந்திருஞ் சூலை"

"அந்தணர் கற்புமாத ரருளிய சாபத்தாலு
முந்திய வினையினாலு முதிரி கர்ப்பமேகத்தாலுஞ்
சிந்தையிற் கொடுமையாலுந் சிவகுரு நிந்தையாலும்
தொந்தமாம் வியாதியாலும் தோன்றிடுஞ் சூலைதானே"

-தேரையர் வாகடம்- பக்கம்333

- Intercourse after oil bath
- Semen arrest
- Excessive intake of Sour and Astringent diets
- Curse by a Chaste women
- Karma, the result of deeds in past birth
- Venereal diseases
- Disrespect to Guru

e) Jeevaratchamirtham (Sapapathi muthaliyar)

- Depression
- Exhaustive running
- Anxiety
- Intake of unpurified Mercurial drugs.
- Excessive consumption of astringent diets
- Indulgence in excessive intercourse
- Excessive starvation.

CLASSIFICATION:

The types of *Soolai* is named differently by many Siddhars. They are as follows,

Yugimuni's view in Yugi vaithiya Sinthamani 800:

Yugimuni classified the *Soolai* in to fifteen types. They are

1. Mega Soolai
2. Muri Soolai
3. Vatha Soolai
4. Pitha Soolai
5. Iyya Soolai
6. Aamma Soolai
7. Ukkara Soolai
8. Gunma Soolai
9. Ularthu Soolai
10. Nithamba Soolai
11. Karai Soolai
12. Sura Soolai
13. Pakka Soolai
14. Karppa Soolai
15. Dhoora soolai

THEERUM AND ELITHIL THEERATHAVAI:

- **Theerum Soolai :**
  1. Dhoora soolai
  2. Mega Soolai
  3. Vatha Soolai
  4. Pitha Soolai
  5. Ularthu Soolai
  6. Karai Soolai
  7. Sura Soolai
  8. Pakka Soolai
  9. Karppa Soolai

- **Elithil Theeratha Soolai :**
  1. Muri Soolai
  2. Iyya Soolai
  3. Aamma Soolai
  4. ***Ukkara Soolai***
  5. Gunma Soolai
  6. Nithamba Soolai

In **Jeevaratchamirtham** classification, therum therathavai of Soolai is same as in Yugivaithiya sinthamani.

Thirumoolar's view:

The Thirumoolar classified the *Soolai* into six types and these are further subdivided into eighteen types. They are as follows,

"ஆறுவகைச் சூலையில் அறுமூன்று பேர்சொல்வார்
வேறு மிதையேல் வெவ்வேறு மொன்றுமில்லை
கூறும் இதன்நாமம் கோடி குணஞ்சொல்வார்
பேறு மறுசூலைப் பேர்சொன்னார் நந்தியே"
-திருமூலர் குணவாகடம்- பக்கம்-32

1. Vatha Soolai
2. Pitha Soolai.
3. Kapha Sool
4. Vatha pitha Soolai
5. Iyya pitha Soola
6. Iyya vatha Soolai

Anuboga vaithiya deva ragasiyam:.

The Soolai is classified in of seven types. They are

1. Vatha Soolai
2. Pitha Soolai
3. Kapha Soolai
4. Thiri Soolai
5. Aamma Soolai
6. Surkkara Soolai
7. Gunma Soolai
8.

Ukkar soolai is one among the fifteen type soolai noi.

CLINICAL FEATURES [NOI KURIGUNANGAL]:

The Clinical features of *Ukkara soolai* according to **Yugimuni in yugivaithiya sinthamani 800:**

Ì òÐÓì ¸¡Ã Ý ¨ÄÂ¢ý Ì ½ó¾¡ý
§¸¡÷¨ ÅÂ¡ö Å¢Ä¡Å¾É¢ø ÓÐ¸¢ø ¦¿ï º¢ø
« ò¾¢Â¢ø ¿¡À¢Â¢É¢ ÄÀ¡É Ì ¾ò¾¢ø
« ¾¢¸òÐý Á¡í¸¢„ó¾¡ý ÅÇ÷óÐ §ÁÅ¢ô
ÀòÐÁ½ü ÀÕì ¨¸§À¡ü ºÄòÐ Å¡Ã
À¾¢ ¦¿Õì¸¢ ã ò¾¢ÃÁ¡íì ¸¢¡¢Îº¢Ô ñ¼¡öò
¾òÐº¼íì ¸Îô¦ÀÎòÐ Á¾¢¸Äí¸¢ò
¾Ç÷îº¢¦Â¡ Î ÁÂì¸Á¡öò ¾û Ùó¾¡§É

--யூகி வைத்திய சிந்தாமணி 800 Àì¸õ-68

Clinical features of *Ukkara soolai*

- Lower abdominal pain
- Abdominal distension
- Pricking pain present in the lower abdomen
- Some abnormal growth developed in lower abdomen and it is compress the urinary tract.
- Burning micturation.
- Frequency of urine.

The above symptoms that are present in other diseases are as follows:

1. **MOOTHIRA KIRICHARAM:**

According To Therayar Vaagadam:

ã ò¾ÃÌ ¸¡¡Î º¡Ì Ì ½ í ì § ¸Ç÷ Ó Î Ì ó ÐÇ¢Â¡ö Å¢Æ ï î º ü§È
¬ üÈ¢ò à Ãõ ¿¼ì ¸ ¦Å¡ð¼¡ ¾Ú ¨ Å ÁÕó¾¡ Ä¡üÚÅ¢ Ì ó
à üÈ¢ Å¢ ¨ ÇÅ¡ö Å¢ ¨ Çó¾ÕÓó¾¡ø ¾ ¨ ¼Â¡ø ¸ Î ¸¢
Á¡üÈ¢ÁÚ ì ¸ Å ¨ ¸ ì ¸¡ ½ ÁÛ §Å¡÷ì ¦ ¸Ä¢É Ó ¨ ÃôÀ¢§Ã.

-§¾Ã ý Å¡ ¸¼õ. - 168

- Drippling of urine
- The person cannot walk far distance due to frequency of urination.
- And it may be cured in surgical treatment also.

2. **VAATHA SOOLAI:**

According To Gunavaagada Thirattu:

¿¢ ¨ ÄÂ¡ É Å¡¾ò¾¢ø ¿¢ ý È¢ Î ï Ý ¨ Ä ¾¡ ý
Ý ¨ ÄÂ¡ É ¨ ¸ ì ¸¡ø Ì ¨ Çî º ¦ ÄøÄ¡ õ Ò ñ §À¡ø
¸ ¨ ÄÂ¡Á ¦ ÄòÐ ì ¸ ñ ¸ ½ ò¾¢ Î í ì ¸¡ú Å¡§Â¡ Î
¾ ¨ ÄÂ¡ É §ÁÉ¢ ¾¡ ý þ ¨ Çò ¾¢ Î §Á..

- Ì ½ Å¡ ¸¼ò ¾¢Ãð Î Àì ¸ õ -73

- Pricking pain is present in extremities
- It affects the “Abanan”. That is, it also affects Micturation and Defication.

3. AZHAL NEER SURUKKU:

¾¡ý ãò¾Ã Á¢¸îº¢ Åì Ìó
¾Ç÷óÐ§Á ¨¸¸¡Ö Á°¾¢Â¡ Ìõ
À¡ÉÕì¸¢îº¢Ú¸Ä¡ ÂÕÅ¢À¡Ôõ
À¡ÃÁ¡ íì Ì¾Á ñ¼Á¢Ä¢í¸ó¾¡Ûíì
¸¡ýÅ¡Ô¾¡ýÁÈ¢ÅÂ¢ÚÓôÒõ
¸Ç¸¦Çý Ú þ¨ÃîºÄ¡öì¸ºí¸¢§ÂÚõ
§Åû¸¡Ô Å¡ÔÄ÷óÐ Á¢Ãðº¢Â¡ Ìõ
Å¢Îõ À¢ò¾ ãò¾Ãò¾ý Å¢ÅÃó¾¡§É.

- ä¸¢¨Åò¾Âº¢ó¾¡Á½¢- 431

- Burning micturation
- Tiredness of upper and lower limbs
- Oliguria
- Pricking pain is present in genital area and anal region.
- Abdominal discomfort
- Dryness of mouth

MUKKUTRA VAERUPADUGAL :(Pathogenesis)

Disease occurs due to the derangement in

- Uyir thathukkal
- Udalthathukkal
- kala marupadu(seasonal changes)
- Thinai(living lands) and
- Udal vanmai.

UYIR THATHUKKAL:

Mukkutra Iyal :

The function of the three uyir thathus:

a) **Vali – (Kattru + Veli)**

b) **Azhal – (Thee)**

c) **Iyyam – (Neer+Mann)**

The alteration of three thathu in their reaction to extrinsic or intrinsic factors results in disharmony. This altered harmony and balance variation of the three thathus results in disease. Their natural ratio (1 :½:¼) to each other is discerned by the physician at the wrist and each nadi is individually assessed for its strength, speed and regularity.

The following poem describes the origin of three Uyir Thathus

"இருப்பான நாடி எழுபதோடீரா
யிரமான தேகத்தில் ஏலப் -பெருநாடி
ஒக்கத் தசமத் தொழிலை யூக்க தசவாயுக்கள்
தக்கபடியானதே சார்பு"

"சாருந் தசநாடி தன்னில் மூலம் மூன்று
பேருமிடமி பிங்கலையும் பின்னலுடன்- மாறும்
உரைக்கவிரற் காற்றொட்டுணர்த்து மேநாசி
வரைச்சுழி யோமையத்தில் வந்து"

"வந்தகலை மூன்றில் வாய்வாமபானனுடன்
தந்த பிராணன் சமானனும் சந்தமுறக்
கூட்டுறவில் ரேசித்தல் கூறும் வாதம் பித்தம்
நாட்டுங்கபமே யாம் நாடு"

- கண்ணுசாமியம்- பக்கம் 36

The three Thathus are manifested at the wrist and are individually and collectively assessed. These three humour are divided in to various types and have their functions specifically.

FUNCTIONS OF VALI:

"ஒழுங்குடள் தாதேழ்மூச் சோங்கி இயங்க
எழுச்சிபெற எப்பணியும் ஆற்ற - எழுந்கிரிய
வேகம் புலன்களுக்கு மேவச் சுறுசுறுப்பு
வாகளிக்கும் மாந்தர்க்கு வாயு"

- மருத்துவ தனிப்பாடல் பக்கம்12

According to the physiological function, vali is ten types. They are

S.NO	VATHAM	GENERAL FEATURES	CHANGES IN UKKARASOOLAI
1.	Piranan(Uyir Kaal)	Responsible for respiration and it is necessary for proper digestion	Normal
2.	Abanan(Kizhnokkumkaal)	Responsible for all downward forces such as voiding of urine, stools, semen, menstrual flow	Affected
3.	Viyanan(paravukaal)	Dwells in the skin and is concerned with the sense of touch... extension and flexion of the parts of the body and distribution, of the nutrients to various parts of the body	Affected
4.	Uthanan (melnokkukaal)	Responsible for all kinds of upward motion such as nausea, vomiting etc...	Normal
5.	Samanan(nadukkaal)	Considered essential for proper digestion, assimilation and carries the digested nutrients to each and every organ	Normal
6.	Nagan	Helps in opening &closing of eyelids	Normal

7.	Koorman	Responsible for vision, lacrimation and yawning	Normal
8.	Kirugaran	Induces appetite, salivation, all secretions in the body including nasal secretion and sneezing	Normal
9.	Devathathan	Induces and stimulates a person to become alert, get anger, to quarrel, to sleep etc	Affected
10.	Dhananjeyan	Resides in the cranium and produces bloating of the body after death. This leaves from the body after 3days of death, forming a way through the skull.	Normal

In Ukkara soolai, Abanan, Viyanan and Devathathan will be mainly affected.

FUNTIONS OF AZHAL:

"பசிதாகம் ஓங்கொளிகண் பார்வைபண் டத்து
ருசிதெரி சத்தி வெம்மை வீரம் - உசித
மதிகூர்த்த புத்திவனப் பளித்துக் காக்கும்
அதிகாரி யாங்கா னழல்"

\- மருத்துவ தனிப்பாடல் பக்கம்16

Azhal is functionally divided in to five types. They are

S.NO	PITHAM	NORMAL FEATURES	CHANGES IN UKKARASOOLAI
1.	Anarpitham(Akkuanal)	Peps up the appetite and aids in digestion.	Normal
2.	Ranjagapitham(Vanna eri)	Responsible for the color and contents of blood.	Normal
3.	Sathagapitham(Attralangi)	Controls the whole body and is held responsible for fulfilling a purpose.	Affected
4.	Pirasagapitham(Ollolithee)	Dwells in the skin and concerned with the shine, glow, texture and its complexion	Normal
5.	Alosagapitham(Nokku Azhal)	Responsible for the perception of vision.	Normal

In Ukkara soolai, sathaga pitham will be affected.

FUNTIONS OF IYAM:

"திடமீயு மென்பிணைப்புத் திண்மையுற்ற யாப்பும்
அடலேர் வழுவழுப்பும் ஆக்கைக் - கிடர்க்கு
வெருவாப் பொறுமையும் மேலான காப்பாம்
பெருமைத்தா மையமெனப் பேசு"

- மருத்துவ தனிப்பாடல் பக்கம்20

It is of five types. They are

S.NO	KABHAM	GENERAL FEATURES	CHANGES IN UKKARASOOLAI
1.	Avalambagam(Alli Iyyam)	Lies in the respiratory organs, exercises authority over other khapas and controls the heart and circulatory system.	Normal
2.	Kilethagam(Neerpi Iyyam)	Found in stomach as its seat, moistens the food, softens and helps to be digested.	Normal
3.	Pothagam(Suvaikanna Iyyam)	Hold responsible for the sensory perception of taste.	Normal
4.	Tharpagam(Niraivu Iyyam)	Presents in the head and is responsible for the coolness of the eyes, sometimes may be referred to as cerebrospinal fluid	Normal
5.	Santhigam(Ondri Iyyam)	Necessary for the lubrication and the free movements of joints.	Normal

UDAL KOORUGAL (SEVEN PHYSICAL CONSTITUENTS):

"இரமிரத் தந்தசை நெய் நிணமென்பு மச்சைவீந்தென்றேழும் முறையே"
சரதமொடு மெய்மனத்து நிறைவுதரும்உயிருட்டுத்தாங்கி யிருக்கும்
உரமுதவும் மேடுபள்ளம் நிரவும் நெய்ப் பசையூட்டும் ஓங்கி நிறுத்தும்
பரந்தென்பின் துளைகடொறும் நிரம்பிடுங்கள் முளைதோன்றப் பண்ணும் தெரிவாய்"
-சித்த மருத்துவாங்கச் சுருக்கம் -பக்கம்334

The human body is made of seven basic physical constituents. These constituents should be in harmony and function normally. Any variation in them will lead to their functional deviations.The Natural characters of the seven physical constituents

S.NO	UDAL KATTUGAL	GENERAL FEATURES	CHANGES IN UKKARASOOLAI
1.	Saaram (digestive essence)	Responsible for the growth& development. It keeps the individual in good temperament and it enriches the bood.	Normal
2.	Senneer (blood)	Responsible for the colour of blood and for the intellect, nourishment, strength, vigour and valour of the body.	Normal
3.	Oon (muscle)	Gives lookable contour to the body as needed for the physical activity. It feed the fat next day and gives a sort of plumpness to the body	Affected
4.	Kozhuppu (fat)	Lubricates the organs to facilitate frictionless functions.	Affected
5.	Enbu (bones)	Supports & protects the vital organs, gives the definite structure of the body and responsible for the posture and movements of the body	Normal
6.	Moolai (bone marrow)	Nourishes the bone marrow and brain which is the centre that controls other systems of body	Normal
7.	Sukkilam/ Suronitham(sperm/ ova)	Responsible for reproduction	Normal

THE VARIATIONS OF THE PHYSICAL CONSTITUENTS:

1. SAARAM

Increased Saaram:Leads to diseases of increased kapham like indigestion Etc

Decreased Saaram : Leads to loss of weight, tiredness, lassitude, dryness of the skin and diminished activity of the sense organs.

2. SENNER

Increased Senner : Causes boils in different parts of the body throbbing pain, anorexia, mental disorder, splenomegaly, Colicky pain., increased blood pressure, reddish eye and Skin, jaundice, haematuria etc.

Decreased Senner : Leads to anaemia, tiredness, neuritis and lassitude, Pallor of body.

3. OON

Increased Oon : Oon in excess causes cervical lymph adenitis, venereal ulcer, tumour in face, abdomen, thigh genitalia etc are the signs of increased Oon

Decreased Oon : Leads to impairment of sense organs, joints jaw, thigh and genitalia gets shortened

4. KOZHUPPU

Increased Kozhuppu: Identical to that of increased Oon associated with Dyspnoea and loss of acidity

Decreased Kozhuppu: Leads to pain in the hip region and diseases of the spleen

5. ENBU

Excess Enbu : Growth in bones and teeth

Decreased Enbu : Loosening of teeth and nails and Splitting and falling of hair

6. MOOLAI

Increased Moolai : Causes heaviness, swollen eyes, swollen phalanges, Oliguria and non healing ulcers

Decreased Moolai : Causes osteoporosis and sunken eyes

7. SUKKILAM / SURONITHAM

Excess Sukkilam/Suronitham : Causes lust towards women and cause Urinary calculus

Decreased Sukkilam/Suronitham : Causes failure in reproduction, pain in the genitalia.

KAALA MARUBADUGAL:

PARUVAKALAM (SEASONS):

According to ancient tamilians, the one year is divided in to six seasons and each season consists of two months and the year starts from Margazhi.

S.NO	KAALAM	TAMIL MONTHS	MUKKUTTRA MARUPAADUGAL
1.	Kaar Kaalam	Aavani & Purattasi Aug 16 To Oct15	*VATHAM*-Vettunilai Vazharchi *PITHAM*-Thanilai Vazharchi
2.	Koothir Kaalam	Iypasi &Karthigai Oct 16 To Dec15	*VATHAM*- Thanilai Vazharchi *PITHAM*- Vettunilai Vazharchi
3.	Munpani Kaalam	Margazhi & Thai Dec16 To Feb15	*PITHAM*- Thanilai Vazharchi
4.	Pinpani Kaalam	Masi& Panguni Feb16 To June15	*KABHAM*- Thanilai Vazharchi
5.	Elavenir Kaalam	Chithirai & Vaikaasi April16 To June15	*KABHAM*- Vettunilai Vazharchi
6.	Mudhuvenir Kaalam	Aani & Aadi June16 To Aug 15	*VATHAM*- Thanilai Vazharchi

THINAI (LAND):

Siddhars classified the lands in to five types. They are

1. Kurunchi - Mountain range
2. Mullai -Pastoral area of the forest
3. Marudham -The fertile river bed
4. Neidhal -The coastal region
5. Paalai - Arid desert

- The winter season gives good health to the man, early summer and latter rainy gives moderate health. Whereas early rainy and latter summer are more prone to diseases, that's why siddhars called it as Aanaga kalam
- Marudha nilam is the fertile area where no disease occurs

RELATION BETWEEN MUKKUTRAM, KAALANGAL AND THINNAIGAL

MUKKUTRAM	PARUVAKALAM(SEASONS)			THINAI
	Thannilai vazharchi (Accumulation)	Vaetrunilai vazharchi (Aggravation)	Thannilai adaithal (Alleviation)	
VATHAM	Mudhuvenil kalam	Kaar kalam	Koothir kalam	Vatha disease is more prevalent in **Neidhal** land
PITHAM	Kaar kalam	Koothir kalam	Munpani	Pitha disease is more prevalent in **Mullai** land
KAPHAM	Pinpani	Elavenil kalam	Mudhuvenil kalam	Kaphadisease is more prevalent in **Kurunchi** land

UDAL VANMAI (IMMUNITY):

Siddhars classify Udal vanmai as three types. They are

1. Iyarkai vanmai
2. Kala vanmai
3. Seyarkai vanmai

Since Ukkara soolai patients are suffering with pain as principal symptom, we came to understand that it is because of alteration in Vali thathu and Vali should be the primary causative factor (Muthanmai kutram). It can be confirmed by the words of great Siddhar Therayer

‘நெடுவாத சார்பதுவுமின்றீ சூலை வராது”

PINIYARI MURAIMAI (DIAGNOSIS):

It means the method of diagnosing the disease.

“மதித்திடற்கருமை வாய்ந்த
மாண்பரிகாரமெல்லாந்
துதித்திட வுணர்ந்தானேனுந்
துகளறப் பணியின்றன்மை
பதித்திட வுணரானாகிற்
பயனுறானாகாலானே
விதித்திடு பிணித்திறத்தை
விளம்புது முதற்கண்மன்னோ”

- சிகிச்சா ரத்தினதீபம்- பக்கம் 3

The above poem describes that diagnosis is very important for the physician to treat the disease.

And,

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Four steps are followed in diagnosing the disease. They are,

a. Poriyaal arithal

b. Pulanal therthal

c. Vinaathal

d. Envagaithervu

In detail,

a. Poriyaal arithal:

In this the physician should carefully observe the changes that occur in the five sensory organs (Porigal) of the patient.

b.Pulanal therthal:

The physician carefully applies his five senses of perception, smell, taste, vision, touch and sound to understand the condition of the patient.

c.Vinaathal:

The physician should interrogate about the patients name, age, occupation,socio economic status, food habits, history of past illness, history of present illness, family history, marital status, menstrual history and frequency of pain.

ENVAGAI THERVUKAL

"நா நிறம் மொழி விழி மலமூத்திரம்
நாடி பரிசமிவை மருத்துவராயுதம்"

-நோய்நாடல் நோய் முதனாடல்-253

¿¡ÊÂ¡ø Ó ý §É¡÷ ¦º¡ ý É ¿ø¦Ä¡Ä¦À¡¦ºò¾¡Öõ
¿¦ÊÂ Å¦Æ¦Â¦É¡Öõ ¿¦ý È ¿¡ì Ì È¦ôÀ¦É¡Öõ
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GENERAL FINDINGS:

1. **NAA:**
    i. Signs and symptoms in the tongue are noted here.
    ii. Color, salivary secretion, ulcers, coating, inflammation, taste changes, deviation and its nature are generally noted.

In *Ukkara soolai* the naa didn't have any impact.

2. **NIRAM:**

The color of the skin is noted here.

In *Ukkara soolai* the niram didn't have any impact

3. **MOZHI:**

Character of the speech is noted, mainly uratha olli(high pitched), thazhntha olli(low pitched), or resembles the sound of any instrument.

In *Ukkara soolai* the mozhi will be affected to the patients who have severe pain leading to the thazhntha olli

4. **VIZHI:**

Character of the eye is noted. Color, Warm, Burning Sensation, Irritation, Visual Perception.

In *Ukkara soolai* the vizhi didn't have any impact.

5. **MALAM:**

The stools are examined for quantity; hardening (malakattu), loose motion (bethi), Color and smell.

In *Ukkara soolai* the malam will be affected in severe enlargement.

6. **MOOTHIRAM:**

A. NEERKURI:

The urine is examined for its color, odour, volume, froth and weight. In *Ukkara soolai* the moothiram will be affected due to urinary micturation.

B.NEIKURI

"அருந்து மாறி ரதமும் அவிரோதமதாய்
அக்கல் அலர்தல்அகாலவூன் தவிர்தழற்
குற்றளவருந்தி உறங்கி வைகறை
ஆடிக்கலசத் தாவியே காதுபெய்
தொருமுகூர்த்தக் கலைக்குட்படு நீரின்
நிறக்குறி நெய்குறி நிருமித்தல் கடனே"

-சித்த மருத்துவாங்கச் சுருக்கம் - பக்கம்509

The early morning urine of the patient is analyzed by dropping a drop of gingely oil on the surface of the urine sample. The accumulation, formations, changes, and dispersal under the sunlight without any external disturbances of the urine sample can be noted.

- Vatha neer - The oil spreads like snake
- Pitha neer - The oil spreads like ring
- Kapha neer - The oil spreads like pearl
- If the oil spreads gradually, it indicates good prognosis
- If the oil spreads fast or gets mixed completely with urine or sinks in urine, it suggests bad prognosis.

Since *Ukkara soolai* is due to the derangement of vatham and pitham,the neikuri will be vatha or pitha neer.

7. NAADI:

Naadi is a Unique Siddha Pulse reading method and it should be felt and not read. Different gaits of Vazhi , Azhal, Iyam like branching, jumping, mixing, rotating and compression can be identified.

NAADI NADAI:

IDENTIFICATION (FINGER)		**INDEX**	**MIDDLE**	**RING**
STRENGTH (IN UNIT)		1	1|2	1|4
PATTERN	**MALE**	Hen	Tortoise	Snake
	FEMALE	Snake	Frog	Swan

"பார்க்கவே பெண்களுக் கிடதுபக்கம்
பதிவாகப் பார்த்திடவே பகரபக்கேளும்
கார்கவே வாதமது சர்ப்பம் போலாய்
கனமான பித்தமது தவளை போலாஞ்
சேர்க்கவே யையமென்ற நாடிதானுஞ்
சிறுநடையா வன்னம் போற் செழிப்பாய்க் கானும்"

-பதிணென் சித்தர் நாடிசாத்திரம் (பரிபூரண நாடி)- பக்கம்2

In Ukkara soolai the Naadi nadai will be as follows;

Vatha naadi:

"வாதமெனும் நாடியது தோன்றிற் சீதம்'...
திரள்வாயு சூலை வலிக்கடுப்புத் திரை"

Kappa pitham:

"காணுமடா சூலையென்றால் சேத்துமபித்தம்"

Vatha kappam:

"பாங்கான வாதத்தில் சேத்துமநாடி...
சேர்ந்தவிடம் வெடிசூலை...."

Pitha vatham:

"சிறப்பான பித்தத்தில் வாதநாடி....
உறைப்பாகச் செரியாமை குன்மஞ் சூலை"

Kappa vatham:

"கண்டாயோ சிலேற்பனத்தில் வாதநாடி....
விடபாகம் விடசூலை பக்கவாதம்"

- சதக நாடி
- நோய்நாடல் நோய் முதனாடல்-253

Vatha pitham:

"திருத்தமாம் வாதத்தோடே தமிங்கொடு பித்தஞ் சேரில்
பொருத்துகள் தோறும் நொந்து போதவே பிடிக்குஞ் சூலை"

- குணவாகட நாடி(அகத்தியர்)-பாடல் 26

In Ukkara soolai the Naadi can be vatha naadi, vatha pitham, vatha kappam, pitha vatham, kappa vatham, kappapitham.

8. PARISAM:

Observations as touch, temperature, sensory impairment, masses, nodes, swelling, and texture of the skin, pain, hardness, edematous, and dullness shall be noted.

In *Ukkara soolai* the parisam may be affected in the lower abdomen severe pain.

LINE OF TREATMENT:

MEDICINE:

- **For Treatment:**

I. Velvanga Parpam:

- 65 mg, Twice Daily.
- With Butter.

DURATION OF TREATMENT:

15 days medicine with pathiyam,

Next 15 days pathiyam without medicine.

PATHIYAM AND APATHIYAM:

A. Pathiyam(diet):

Early morning:

A glass of luke warm water mixed with the half of the lime and a spoon of honey.

Diet:

- Rice: Kaar, Kuruvai, Manakathai, Boiled rice (any one)
- Soy and other foods like legumes, tea, apples and onions may help prevent or treat the symptoms.
- **Increase** intake of fruits, vegetables and whole grains, soy, and green tea, foods rich in omega 3 oils (cold-water fish – salmon, sardines, mackerel) and in zinc (raw pumpkin seeds for omega-3 and zinc)

B. Apathiyam(Avoid):

- **Reduce** foods high in fat and cholesterol (butter and margarine, beef and whole milk), sweet foods, and refined carbohydrates (white bread and white-flour pasta)
- **Avoid or decrease** intake of alcohol, coffee, and beer, particularly after dinner, and tobacco.
- Hot and sour tastes
- Kizhangu vagai(tubers), Vatha foods.

5. ADVICE:

1. Some men who are nervous and tense urinate more frequently. Reduce stress by exercising regularly and practicing relaxation techniques such as meditation.

2. When you go to the bathroom, take the time to empty your bladder completely. This will reduce the need for subsequent trips to the toilet.

3. Talk with your doctor about all prescription and over-the-counter medications you take; some may contribute to the problem. Your doctor may be able to adjust dosages or change your schedule for taking these drugs, or he or she may prescribe different medications that cause fewer urinary problems.

4. Avoid drinking fluids in the evening, particularly caffeinated and alcoholic beverages. Both can affect the muscle tone of the bladder, and both stimulate the kidneys to produce urine, leading to nighttime urination.

❖ **Yoga practice**

- Pranayamam
- Sarvangasanam
- Dhanurasanam
- Halasanam
- Yogamuthirai.

6. PREVENTION:

- Balanced & Low fat diet
- Regular exercise
- Suriya namesakaram
- Oil bath twice in a week
- Avoid Junk foods
- Avoid tobacco, Alcohol

Modern Aspects

MODERN ASPECT

Introduction:

Benign Prostatic Hyperplasia is the most common benign tumor in men, and its incidence is age-related. Benign Prostatic Hyperplasia (BPH) is a progressive condition characterised by prostate enlargement accompanied by lower urinary tract symptoms (LUTS). It contributes to, but is not the sole cause of LUTS. It is well known that BPH and the resultant LUTS is very common in elderly men

It was estimated that in the male population. Although clinical evidence of disease occurs less commonly, symptoms of prostatic obstruction are also age-related. At age 55, approximately 25% of men report obstructive voiding symptoms. At age 75, 50% of men complain of a decrease in the force and calibre of their urinary stream.

Consideration of prostatic diseases will be facilitated by a brief introduction to the normal anatomy of the prostate.

Anatomy of the Prostate Gland:

The **prostate** is a Greek word. Prostate literally means "one who stands before", "protector" or "guardian".Which adds secretions to the sperm during the ejaculation of semen.

In 2002, female paraurethral glands, or Skene's glands, were officially renamed the female prostate by the Federative International Committee on Anatomical Terminology.

Shape:

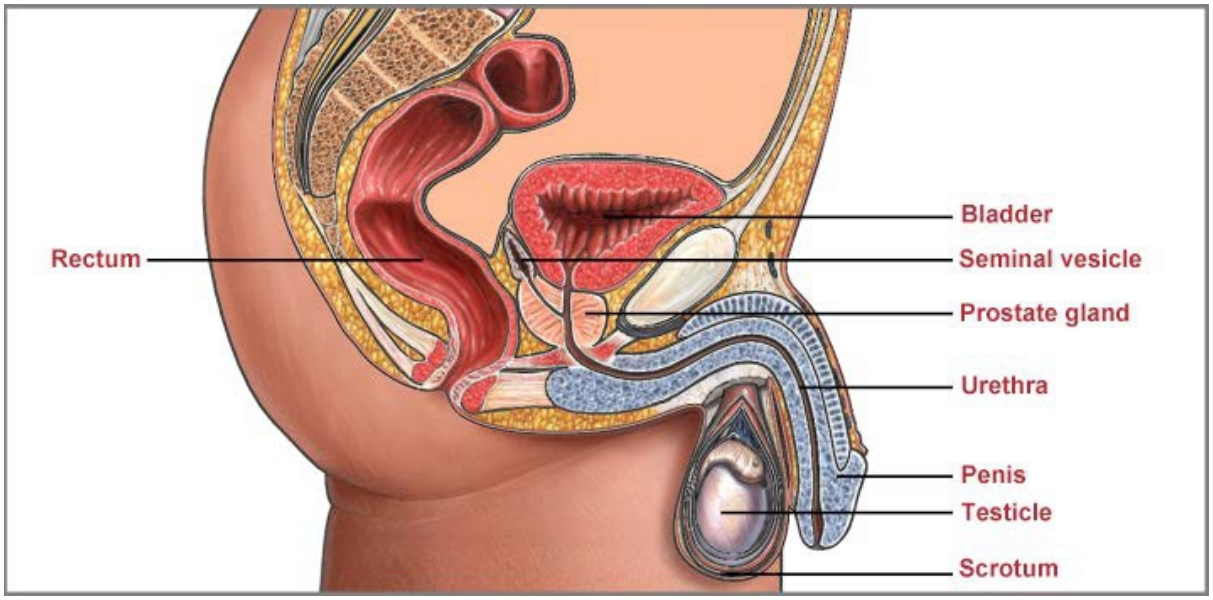
It is a Chestnut-shaped or conical reproductive organ and it is partly glandular and partly muscular body.

Location:

It is situated in the pelvic cavity located directly beneath the bladder in the male. And it is placed immediately below the internal urethral orifice and around the commencement of the urethra.

Development:

The prostatic part of the urethra develops from the *pelvic* part of the urogenital sinus or endodermal origin.



Dimension:

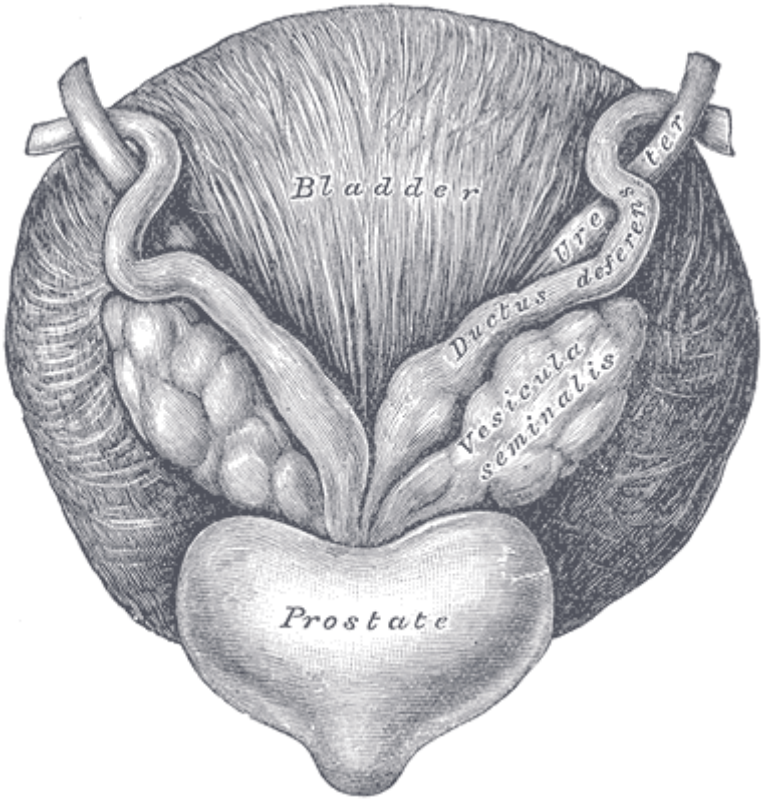
The prostate measures about 4 cm. transversely at the base, 2 cm. in its antero-posterior diameter, and 3 cm. in its vertical diameter. Its weight is about 8 gm.

Features:

It has 2 parts and 4 surfaces.

- A base, an apex,
- An anterior, a posterior and two lateral surfaces.

Base:



The base is directed upward, and is applied to the inferior surface of the bladder, The greater part of this surface is directly continuous with the bladder wall; the urethra penetrates it nearer its anterior than its posterior border.

Apex:

The apex is directed downward, and is in contact with the superior fascia of the urogenital diaphragm.

Surfaces:

Posterior surface:

The posterior surface is flattened from side to side and slightly convex from above downward; it is separated from the rectum by its sheath and some loose connective tissue, and is distant about 4 cm. from the anus. Near its upper border there is a depression through which the two ejaculatory ducts enter the prostate.

This depression serves to divide the posterior surface into a lower larger and an upper smaller part. The upper smaller part constitutes the middle lobe of the prostate and intervenes between the ejaculatory ducts and the urethra, it varies greatly in size, and in sme cases is destitoute of glandular tissue. The lower larger portion sometimes presents a shallow median furrow, which imperfectly separates it into a rightand a left lateral lobe: these form the main mass of the gland and are directly continuous with each other behind the urethra.

In front of the urethra they are connected by a band which is named the isthmus: this consists of the same tissues as the capsule and is devoid of glandular substance.

Anterior surface:

The anterior surface measures about 2.5 cm. from above downward but is narrow and convex from side to side. It is placed about 2 cm. behind the pubic symphysis, from which it is separated by a plexus of veins and a quantity of loose fat. It is connected to the pubic bone on either side by the puboprostatic ligaments. The urethra emerges from this surface a little above and in front of the apex of the gland.

Lateral surfaces:

The lateral surfaces are prominent, and are covered by the anterior portions of the Levatores ani, which are, however, separated from the gland by a plexus of veins.

The prostate is perforated by the urethra and the ejaculatory ducts. The urethra usually lies along the junction of its anterior with its middle third. The ejaculatory ducts pass obliquely downward and forward through the posterior part of the prostate, and open into the prostatic portion of the urethra.

Structure:

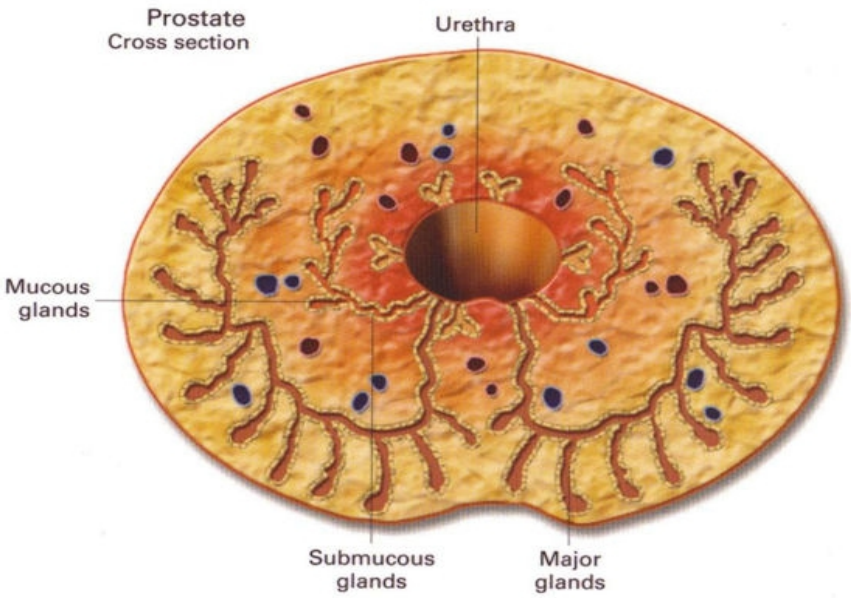
The gland surrounds the urethra, the duct that serves for the passage of both urine and semen, rounded at the top, the gland narrows to form a blunt point at the bottom, or apex.

The prostate is immediately enveloped by a thin but firm fibrous capsule, distinct from that derived from the fascia endopelvina, and separated from it by a plexus of veins.

This capsule is firmly adherent to the prostate and is structurally continuous with the stroma of the gland, being composed of the same tissues, viz., non-striped muscle and fibrous tissue. The substance of the prostate is of a pale reddish-gray color, of great density, and not easily torn. It consists of glandular substance and muscular tissue.

Muscular tissue:

The muscular tissue according to Kölliker, constitutes the proper stroma of the prostate, the connective tissue being very scanty, and simply forming between the muscular fibers, thin trabeculæ, in which the vessels and nerves of the gland ramify. The muscular tissue is arranged as follows, immediately beneath the fibrous capsule is a dense layer, which forms an investing sheath for the gland.Secondly, around the urethra, as it lies in the prostate, is another dense layer of circular fibers, continuous above with the internal layer of the muscular coat of the bladder, and blending below with the fibers surrounding the membranous portion of the urethra.



Between these two layers strong bands of muscular tissue, which decussate freely, form meshes in which the glandular structure of the organ is imbedded. In that part of the gland which is situated in front of the urethra the muscular tissue is especially dense, and there is here little or no gland tissue; while in that part which is behind the urethra the muscular tissue presents a wide-meshed structure, which is densest at the base of the gland that is, near the bladder becoming looser and more sponge-like toward the apex of the organ.The prostate gland is a conglomerate of 30 to 50 tubular or saclike glands that secrete fluids into the urethra and ejaculatory ducts. The secretary ducts and glands are lined with a moist, folded mucous membrane. The folds permit the tissue to expand while storing fluids. Beneath this layer is connective tissue composed of a thick network of elastic fibres and blood vessels. The tissue that surrounds the secretary ducts and glands is known as , this contains muscle, elastic fibres, and collagen fibres that give the prostate

gland support and firmness. The capsule enclosing the prostate is also of interstitial tissue.

Glandular substance :

The **glandular substance** is composed of numerous follicular pouches the lining of which frequently shows papillary elevations. The follicles open into elongated canals, which join to form from twelve to twenty small excretory ducts. They are connected together by areolar tissue, supported by prolongations from the fibrous capsule and muscular stroma, and enclosed in a delicate capillary plexus. The epithelium which lines the canals and the terminal vesicles is of the columnar variety.

The prostatic ducts open into the floor of the prostatic portion of the urethra, and are lined by two layers of epithelium, the inner layer consisting of columnar and the outer of small cubical cells. Small colloid masses, known as **amyloid bodies** are often found in the gland tubes.

Vessels and Nerves:

The **arteries** supplying the prostate are derived from the internal pudendal, inferior vesical, and middle hemorrhoidal. Its veins form a plexus around the sides and base of the gland; they receive in front the dorsal vein of the penis, and end in the hypogastric veins. The **nerves** are derived from the pelvic plexus.

Function:

The function of the prostate is to secrete a slightly acidic fluid, milky or white in appearance, that usually constitutes 50–75% of the volume of the semen along with spermatozoa and seminal vesicle fluid. Semen is made alkaline overall with the secretions from the other contributing glands, including, at least, the seminal vesicle fluid. The alkalinity of semen helps neutralize the acidity of the vaginal tract, prolonging the lifespan of sperm. The alkalinization of semen is primarily accomplished through secretion from the seminal vesicles.

The prostatic fluid is expelled in the first ejaculate fractions, together with most of the spermatozoa. In comparison with the few spermatozoa expelled together with mainly seminal vesicular fluid, those expelled in prostatic fluid have better motility, longer survival and better protection of the genetic material.The prostate also contains some smooth muscles that help expel semen during ejaculation.

Secretion:

Prostatic secretions vary among species. They are generally composed of simple sugars and are often slightly acidic.

In human prostatic secretions, the protein content is less than 1% and includes proteolytic enzymes, prostatic acid phosphatase, beta-microseminoprotein, and prostate-specific antigen. The secretions also contain zinc with a concentration 500–1,000 times the concentration in blood.

Female prostate gland:

The Skene's gland, also known as the paraurethral gland, found in females, is homologous to the prostate gland in males. However, anatomically, the uterus is in the same position as the prostate gland. In 2002 the Skene's gland was officially renamed to female prostate by the *Federative International Committee on Anatomical Terminology*.

The female prostate, like the male prostate, secretes PSA and levels of this antigen rise in the presence of carcinoma of the gland. The gland also expels fluid, like the male prostate, during orgasm.

The only three pathological processes that affect the prostate gland. There are Inflammation, Benign enlargement, and tumors. Of these three, the benign prostatic enlargement(BPH) are by far the most common and occur so often in advanced age that they can almost be constructed as a “normal” ageing process.

Benign Prostatic hyperplasia

Introduction:

The noncancerous enlargement of the prostate called Benign prostatic hyperplasia (BPH). It affects nearly all men reaching normal life expectancy. BPH is common in older men. By the age 60, more than half of men have BPH. By the age 85, about 90% of men have BPH, but only 30% of men will be bothered by their symptoms.

BPH affects the inside part of the prostate first. Enlargement frequently causes a gradual squeezing of the urethra where it runs through the prostate.

Sometimes this causes difficulty in urinating or other urinary problems. BPH generally does not interfere with sexual functioning.

BPH is not cancer nor does it lead to cancer. However , it is possible for a men to have both BPH and prostate cancer.

Definition:

BPH is a noncarcenogenic (benign) growth of the cells within the prostate gland.

From 40 years of age the prostate increase in volume by 2.4 cms qube per year on average. The process begins in the periurithral (tranitional) zone and involves both glandular and stromal tissue to variable degree. Associated symptoms are common from 60 years of age and some 50% of men over 80 years will have lower urinary tract symptoms (LUTS) associated with BPH.

Prevalence:

The prevalence of clinical BPH is approximately 70% substantially greater than that of other common diseases such as a diabetes or asthma.

The prevalence of BPH increase with age.

According to the National Institutes of Health, there are more than 7.8 million BPH diagnoses made.

Histological evidence of BPH emerges after age 30, with 50% prevalence in men age 50-61 and 90% prevalence by age 90. However, it is difficult to predict how many of these cases will progress to clinical BPH. The overall prevalence of clinical BPH (BPH with LUTS) is 10.3%, with a maximum prevalence of 24% by age 80. It is estimated that 45% of nonsymptomatic 46-year-old men with histological BPH will develop LUTS over the next 30 years.

Causes:

The cause of BPH is not well understood. No definite information on risk factors exists.

For centuries, it has been known that BPH occurs mainly in older men and that it doesn't develop in men whose testes were removed before puberty. For this reason, some researchers believe that factors related to aging and the testes may spur the development of BPH.

Throughout their lives, men produce testosterone, an important male hormone, and small amounts of estrogen, a female hormone. As men age, the amount of active testosterone in the blood decreases, leaving a higher proportion of estrogen.

Studies done on animals have suggested that BPH may occur because the higher amount of estrogen within the gland increases the activity of substances that promote cell growth.

Another theory focuses on dihydrotestosterone (DHT), a substance derived from testosterone in the prostate, which may help control its growth. Most animals lose their ability to produce DHT as they age. However, some research has indicated that even with a drop in the blood's testosterone level, older men continue to produce and accumulate high levels of DHT in the prostate. This accumulation of DHT may encourage the growth of cells.

Scientists have also noted that men who do not produce DHT do not develop BPH.

Some researchers suggest that BPH may develop as a result of “instructions” given to cells early in life. According to this theory, BPH occurs because cells in one section of the gland follow these instructions and “reawaken” later in life. These “reawakened” cells then deliver signals to other cells in the gland, instructing them to grow or making them more sensitive to hormones that influence growth.

Enlargement of central part of the prostate (transition zone) narrows the calibre of the urethra passing through its middle and causes obstruction to the flow of urine from the bladder.

Clinical features:

The primary symptoms of BPH are due to the prostate obstructing the urethra. They consist of,

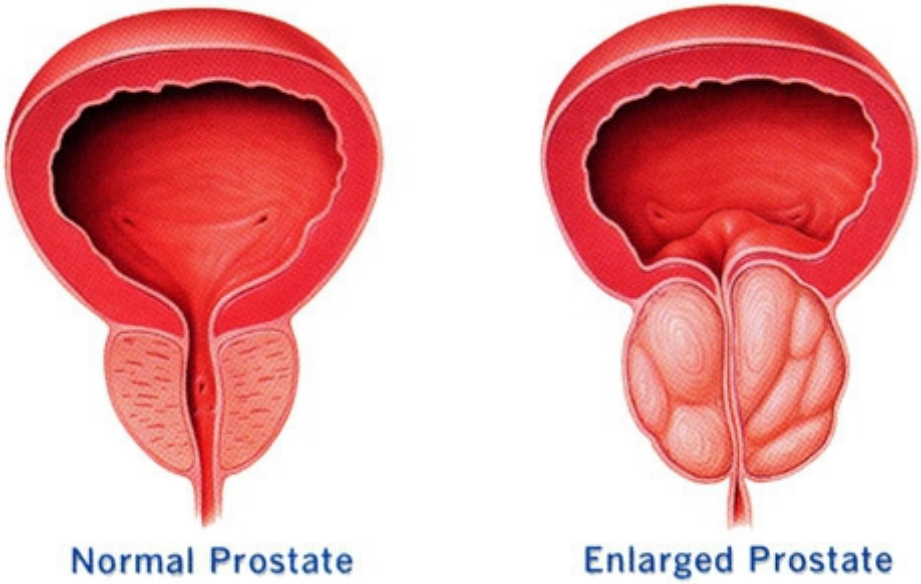
- Hesistancy,
- Poor prolonged flow
- And a sensation of incomplete empting,

Secondary (irritative) symptoms ,

- Comprising urinary frequency (going often),
- Urgency of micturation(going in a hurry)
- And urge incontinence (leaking if you can’t get to a toilet in time).

Urge incontinence are not specific to BPH.

Patients may present more dramatically with acute urinary retention when they are suddenly unable to micturate and develop a painful distended bladder. This is often precipitated by excessive alcohol intake, constipation or prostatic infection. It is an emergency and requires the bladder to be drained by catheter to receive the retention.



In chronic urinary retention the bladder slowly distends due to inadequate emptying over long period of time. This condition is characterized by pain-free bladder distention which may result in hydroureter, hydrinephrosis and renal failure.

Patients with chronic retention can also develop acute retention; so called acute on chronic retention. They require careful management because of their renal failure.

In some patients, the back-pressure caused by the obstruction separates nerve endings from the bladder muscle fibres they are travelling towards, causing the bladder to behave in a reflex manner rather like a baby's bladder does.

Other **BPH symptoms** relate to the stagnation of urine, which can lead to urinary infection (pain on and frequency of passing urine) or stone formation (recurent urinary infections and frequency), and the symptoms of kidney failure.

Getting up at night once or twice to pass urine (‘nocturia’) becomes increasingly common with increasing age in both sexes as the kidneys make more urine at night. It is not necessarily a symptom of problems, although it can be bothersome.

Dribbling urine after the main stream has finished is common in middle-age and beyond and is also not necessarily a sign of prostate or bladder problems. It is due to pooling of urine in the ‘U’ bend of the urethra after it has left the bladder due to age-related weakness of the muscle (‘bulbospongiosus’) that surrounds that part of the urethra.

This is the same muscle that contracts to expel semen out of the urethra during climax. The solution is either to push a finger up behind the scrotum and run it forwards to milk the urine out of the urethra or simply to put some toilet tissue into your underwear.

Investigations:

All patients with bothersome urinary symptoms should be investigated as the severity of BPH symptoms does not always reflect the severity of the problem.

Urine sample:

This is taken to look for the presence of blood or inflammatory cells in the urine and to exclude infection.

1. Albumin
2. Sugar
3. Deposit

Blood tests:

Prostate-specific antigen (PSA):

PSA is a chemical which is only produced by prostate cells and which is detectable in the blood. A number of factors cause its increased production:

1. Increasing age,
2. Increasing prostate size,

3. Ejaculation,
4. Urinary infection,
5. Prostatitis,
6. Prostate injury (such as a biopsy)
7. Prostate cancer.

PSA, a single chain glycoprotein with a molecular weight of 34 kilodaltons, is produced by all prostatic epithelial cells whether benign or malignant. Serum PSA levels are believed to be elevated through leakage of intraprostatic PSA into the systemic circulation. In the systemic circulation, PSA occurs in both a free form (free PSA) and a form complexed to endogenous prostate inhibitors such as alph-1-antichymotrypsin. A common, reliable, reproducible test that is easy to obtain in nearly any clinical setteing, serum PSA is the biomarker available for prostate cancer.

Flow rate (FR) :

Flow rate is a simple test performed to determine how quickly a bladder can be emptied and is used to quantify the presence or absence of obstruction, which may indicate BPH. With a comfortably-full bladder, the patient urinates into a machine which measures the rate of urine flow. A ‘bell’-shaped curve with a maximum flow of 15 ml/sec is normal.

Post-void residual (PVR):

Post-void residual test measures the amount of urine that remains in the bladder by means of an ultrasound scan, usually after a flow rate. Up to 100 ml is normal.

Ultra Sonogram:

Estimate the dimension and volume of the prostate gland.

Digital Rectal Examination (DRE):

This examination is usually the first test done. The doctor inserts a gloved finger into the rectum and feels the part of the prostate next to the rectum. This examination gives the doctor a general idea of the size and condition of the gland.

IPSS (International Prostate symptoms Score):

The IPSS is a questionnaire designed to determine the seriousness of a man's urinary symptoms and to help diagnose BPH. The patient answers seven questions related to common symptoms of BPH. How frequently the patient experiences each symptom is rated on a scale of 1 to 5.

These numbers added together provide a score that is used to evaluate the condition. An IPSS score of 0-7 means the condition is mild; 8-19, moderate; and 20-35, severe

International Prostate Symptom Score (IPSS)

Name:_____ Date:_____

Age: 40-49 50-59 60-69 70+

	Not at all	Less than 1 time in	Less than half the	About half the time	More than half the	Almost always	Your score
Incomplete emptying How often have you had a sensation of not emptying your bladder completely after you finish urinating**?**	**0**	**1**	**2**	**3**	**4**	**5**	
Frequency How often have you had to urinate again less than two hours after you finished urinating?	**0**	**1**	**2**	**3**	**4**	**5**	
Intermittency How often have you found you stopped and started again several times when you urinated?	**0**	**1**	**2**	**3**	**4**	**5**	
Urgency How difficult have you found it to postpone urination?	**0**	**1**	**2**	**3**	**4**	**5**	
Weak stream How often have you had a weak urinary stream?	**0**	**1**	**2**	**3**	**4**	**5**	
Straining How often have you had to push or strain to begin urination?	**0**	**1**	**2**	**3**	**4**	**5**	

	None	1 time	2 times	3 times	4 times	5 times or more	Your score
Nocturia How many times did you typically get up at night to urinate?	**0**	**1**	**2**	**3**	**4**	**5**	

OTHERS:

Cystoscopy or telescopic inspection:

Cystoscopy inspection of the bladder is usually performed in patients with ‘irritative’ bladder symptoms to exclude physical bladder irritants, such as a stone or cancer, which are unusual.

It can be performed awake under local anaesthetic and using a flexible telescope or whilst asleep under general anaesthetic.

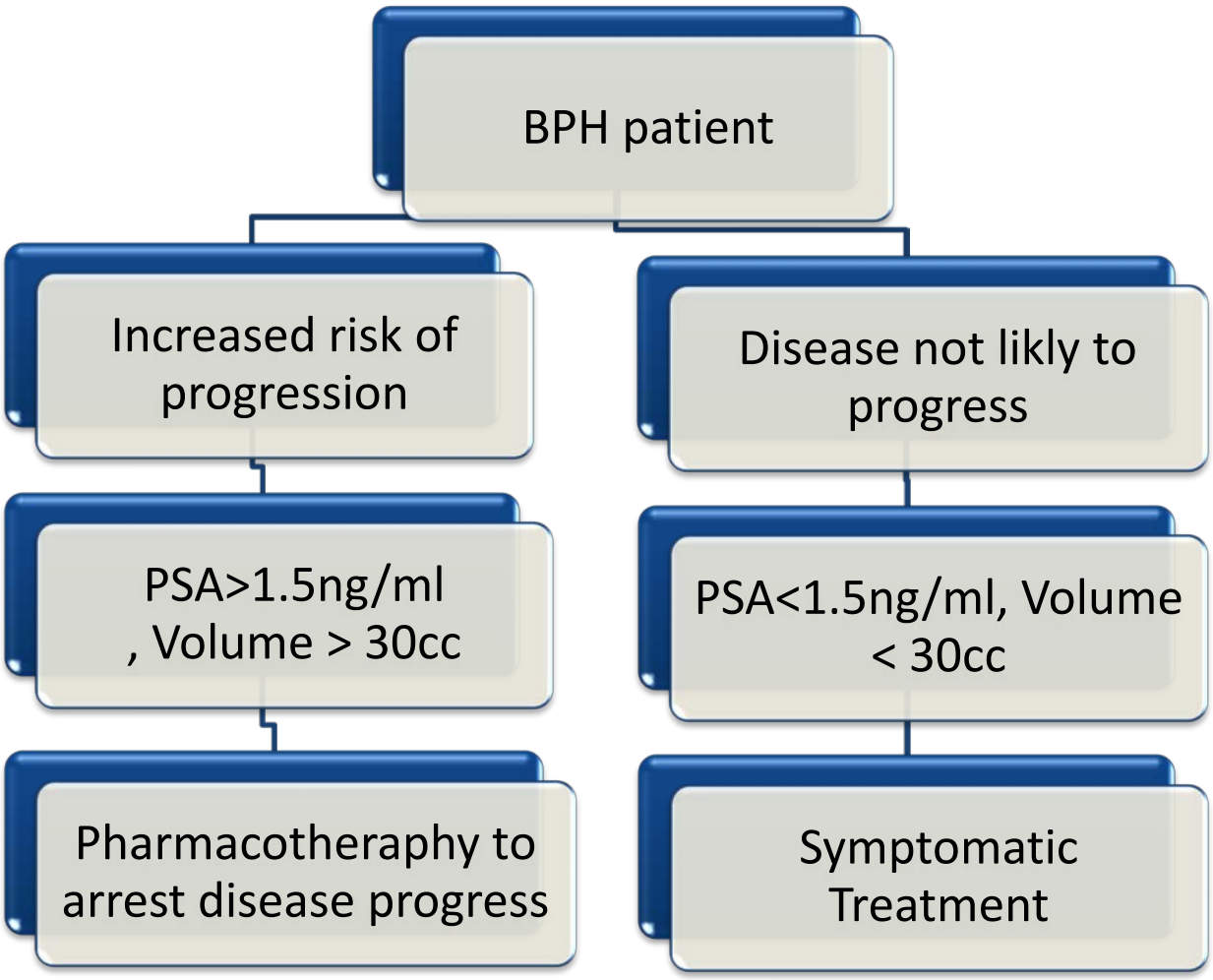
In this examination, inserts a small tube through the opening of the urethra in the penis. This procedure is done after a solution numbs the inside of the penis so all sensation is lost. The tube, called a cystoscope, contains a lens and a light system that help see the inside of the urethra and the bladder. This test allows to determine the size of the gland and identify the location and degree of the obstruction.

Urodynamic test:

Urodynamic testing involves filling the bladder via a catheter inserted through the penis and measuring its behaviour (via a second catheter in the penis and a third in the rectum) as it fills and empties.

It can be an uncomfortable and undignified test and fortunately is not often necessary as other methods exist to obtain the same information in most patients. However, then these tests are equivocal, urodynamics often provide the answer

Disease progress:



Management:

Best Foods for Prostate Health:

Prostrate Health Diet

- **Vegetables from the cruciferous family** (broccoli, cabbage and cauliflower) contain isothiocyanates, which are phytochemicals that appear to be protective.
- **Omega-3** fats seem to reduce the risk of prostate cancer, Use Fish and vegetable oils high in Omega-3 Fats
- Margarine, vegetable oils, nuts and seeds, wheat germ and whole grains are a good sources. **Vitamin E** is recognized to reduce prostate inflammation
- **Whole grains** present fibre, selenium, vitamin E and phytochemicals, all of which play a role in the prevention of cancer.
- Foods such as tomatoes, tomato products, red grapefruits and watermelons appear to reduce the risk of **Prostate Cancer**. Tomato-based pasta sauces and soups may be especially beneficial. Lycopene is fat soluble so is better absorbed when eaten with a little fat.

- **Antioxidant** is found in nuts especially Brazil nuts, seafood, fish, wheat bran, wheat germ, oats and brown rice.
- **Selenium** is another mineral that may offer protection.
- **Soy products** can help prevent prostate enlargement and may slow tumour growth.
- **Red meat** is high in saturated animal fats and has been linked to an increased incidence of prostate problems. **Avoid Red Meat.**
- **Obesity** has also been linked to **prostate troubles** and cutting back on **red meat** can help you **lose weight**.
- Drink **plenty of fluids** to flush the bladder. Caffeine, beer and spices should be reduced to a minimum.

Trial drugs

LITERATURE REVIEW OF TRIAL DRUGS

¦ÅûÅí¸õ :

Chemical Name : Stannum (TIN)

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À¡ : ¸¡÷ôÒ

¦ºö¨¸ :

- ¸ÕÁ¿¡ºÉ (Anti-dote)
- ¾¡Ð ¦ÅôÀ¸üÈ (Sedative)
- ÐÅ÷ôÀ (Astringent)
- Å¦ì¸ÓÕì¸ (Deobstruent)

Ì ½õ (Properties) :

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¸üÈ¡ ¨ Æ:-

Botanical Name : Aloe vera

Family : Lilliaceae

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- ¯ÃÁ¡ì ¸¼ (Tonic)
- ¯¼ü§ÈüÈ¼ (Nutrient)
- ¿¦÷ÁÄõ§À¡ì ¸¼ (purgative)
- ÕÐ× ñ ¼¡ì ¸¼ (Emmenagogue)

Ì ½õ (Properties) :

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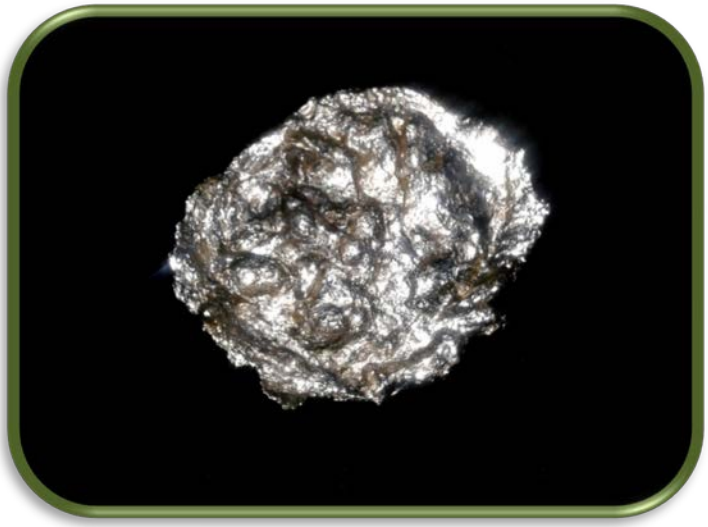
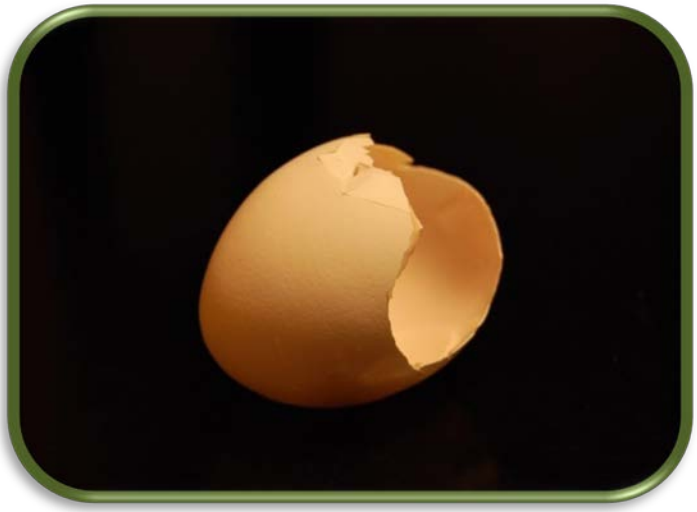
DRUGS OF VELVANGA PARPAM



PURIFIED VELVANGAM



EGG SHELL

ALOVE VERA

Materials & Methods

METERIALS AND METHODS

PROTOCOL

Introduction:

The clinical trial for *benign prostate hyperplasia* was decided to be conducted as an open label study.

Data collection:

- Literary evidence from various
- Siddha books
- Medical journals
- Internet

Trial spot:

The entire study was conducted on patients on attending the OPD of Government Siddha Medical College, Aringnar Anna Hospital Of Indian Medicine Campus , Arumbakkam, Chennai- 106 , during the period 2010-2013.

Population:

The population consists of Benign Prostatic hyperplasia patients satisfying the inclusion and exclusion criteria mentioned below.

Inclusion Criteria:

1. Age: between 50 - 80 years.
2. Clinical signs and symptoms of BPH for $\geq$ 1months.
3. Willing to give specimen of blood for investigation when required.
4. PSA Score < 4ng.
5. Post void residual urine volume $\leq$ 350cc.
6. Willing to attend the OPD once in 7 days.
7. Adhere to protocol requirements with written informed consent.

Exclusion Criteria*:*

1. History/ Clinical evidence of Prostate Cancer or a serum PSA > 4 ng/ml.
2. Clinical evidence of any of the bladder or Urinary tract conditions.
3. Post void residual urine Volume $\geq$ 350 ml by ultrasound.
4. Patient who have participated in a drug study in past 3 months.

Duration of Treatment:

- 30 days.

Patients were followed under the guidance and supervision of the HOD, Professor, Reader, Lecturer and Asst. Lecturer of the Maruthuvam P.G Department, GSMC, Chennai-106.

20 patients were selected and carefully studied for their history, clinical examinations, investigations and management.

Evaluation of Clinical Parameters:

The history includes past, personal, family, occupation, dietary habits, Seasonal history, and associated history.

Investigations:

- **Blood**
  - TC,
  - DC,
  - ESR,
  - HB,
  - Blood sugar,

- Blood Urea
- Serum Cholesterol
- Serum Creatinine

❖ Prostate Specific Antigen (PSA)

❖ **Urine:**

- Albumin
- Sugar
- Deposit

❖ **USG – KUB:**

- **Prostate**
  - Measurement
  - Volume
  - Post void residual urine

Investigations Based On Siddha System*:*

1. *Envagai Thervu:*

   Na, Niram, Mozhi, Vizhi, Sparisam, Naadi, Malam, Moothiram

2. *Neerkuri:*

   Niram, Manam, Eadai, Nurai, Enjal

3. *Neikuri :*

A case sheet format was prepared on the basis of the Siddha methodology example envagai thervvugal, mukkutram, nilam, kaalam, udal thathugal, including neerkuri and neikuri. Individual case sheet was maintained for each patient at outpatient department.

TRIAL DRUG:

Drug:

Velvanga Parpam:

Reference:

Veeramamunivar vagada thirattu

Ingredients:

1. Velvangam (Tin) – 100gm
2. Egg shell – 100gm
3. Aloe vera juice

Procedure:

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ºùÅ¸ýÈ Ä½¼ò§¾¡ Ã¡ÉÐÅ¡õ- þùÅ¾¨ É ì

¸øÅò¾ð Îô¦À¡ÊòÐì ¸¡ Ï ¸Õõ ¦À¡ü¸Äò¾ø

¦ÅûÅí¸õ §À¡ð¼Îôº ÁÐ.

²üÈò ¾ÆÄð ¦¼¡ò¾ ñ ¼ò Ðûºú¸ô

§À¡ð Î ò§¾ öô À¡ö¸¡Â ¦À¡üÚ Îô À¡ø- º¡üÚ à û

¦ºýÈÀýÒ ¿À¡÷ì¸Š ¦º÷ó¾Åí¸õ ÀüÀÁ¡õ

´ýÚ¸Õô À¡ÂÕì Ì §Á¡÷

Á¦ ñ Î í ¸ø Åò¾Ä¦ð Î ¦Áî Í Ì Á ¡¦îº¡üÈ¦ü

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¬ ì ¸¦ ÔÄ÷ò¾ ÂÆ Ì ¦ÀÈ §Å¡ðÊÄ¦ð Î ô

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Ô¸ó¾ ÅÈðÊÂ¦ü¸¡ü§È¡¼ - ¿¦¸úó¾Ò¼õ

§À¡ð ¦¼ Î ì ¸Å¡ ½ Ãò ¨ ¾ô §À¡ ý È ¿¦ÈôÀüÀÁ¡õ

° ð¼Ç× Ì ý È¦Â ¨ Ã ¦Â¡ ý Ú

º¡üÚ Í ¸ ¿÷ô ¦ÀÕ ì Ì ó ¾¡¾ý º¦Ã¡Å¸ Óõ

§¾¡üÈ¦Â¿÷ô ¨ ÀÂ¦ý É Ð Ð÷ÀÄÓõ- § ¸ð ¨ ¼ÔÚõ

§¾¸ÀÄó ¾¡Ð‰Ê §ºÕ ¦Á¡Õ ¾ í ¸¦ø ¨ Ä ¦Â ý

Å¡¸¼ò¾ §ÄÔÚ¾ ¨ Å.

-Å¦ÃÁ¡Ó É¦Å÷ Å¡¸¼ ¾Ãð Î -72

Purified velvangam is taken in a iron bowl and purified powder of egg shell is poured on the melted velvangam and stir it thoroughly until the velvangam completely merge. Add juice of aloe vera and grind it for 12 hours(4 samam) and keep it in pudam.

Dosage:

65 mg

Adjuvant:

Honey / butter.

Duration:

30 days

Indication:

- Normal urine flow
- Urinary Bladder disease.

DRUG

VELVANGA PARPAM

Results & Observations

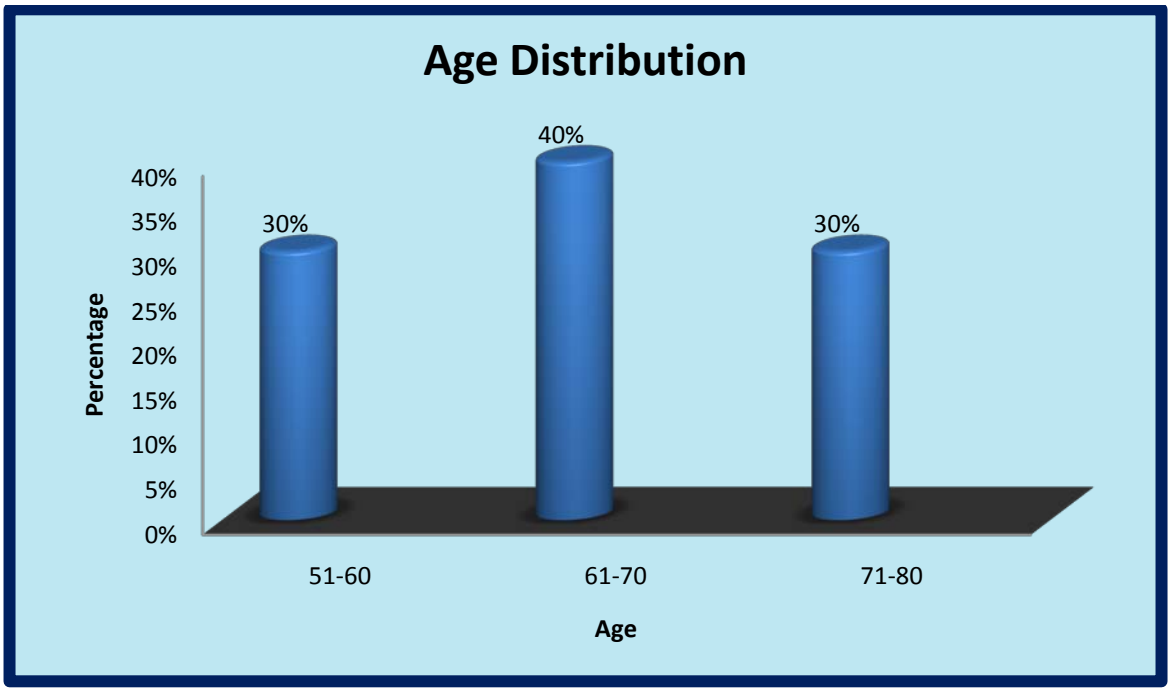
RESULTS AND OBSERVATIONS

The factors considered for the purpose of the study comprised of the following:

- Age Distribution
- Thinai
- Paruvakaalam
- Occupational status
- Socio economic Status
- Food habits
- Personal habits
- Symptoms
- Classifications of results according to Vali, Azhal & Iyyam
- Udal kattugal
- Enn vagai thervu
- Naadi
- Classification on the basis of Neikuri
- Clinical progress
- Results after treatment.

AGE DISTRIBUTION:

SL.NO	AGE	NO. OF PATIENTS /20	PERCENTAGE
1.	51-60	6	30%
2.	61-70	8	40%
3.	71-80	6	30%

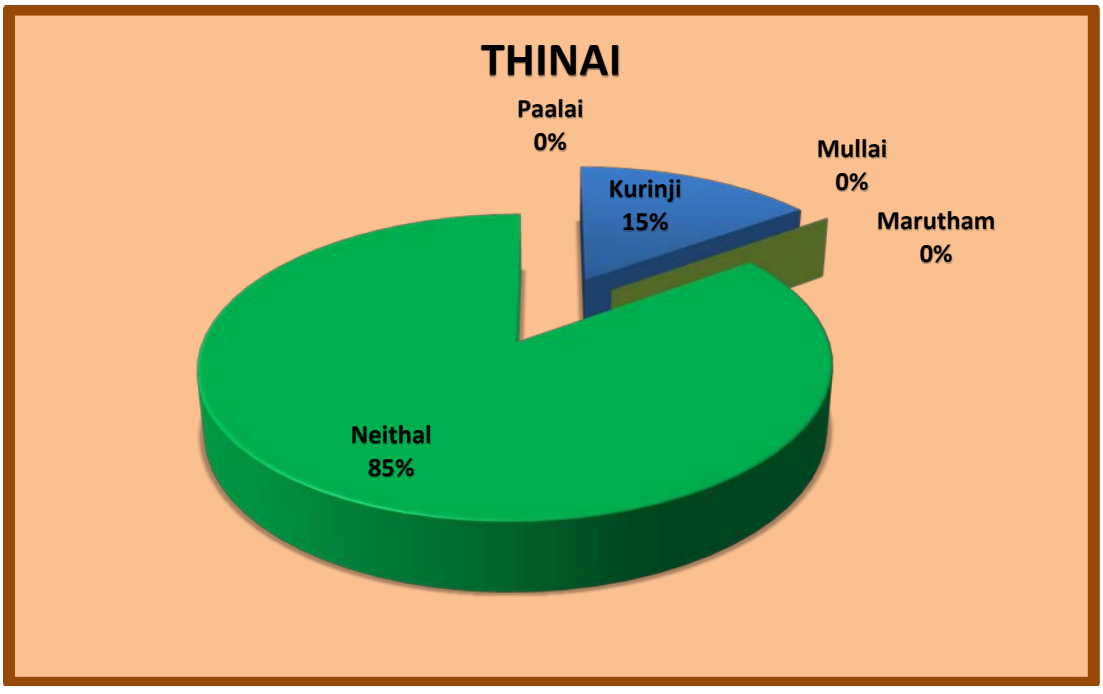


Inference:

According to the above mentioned data 40% of patients were in age groups 61-70 years, 30% of patients were in age group 71-80 year,30% of patients were in age group 51-60 years.

THINAI:

SL.NO	THINAI	NO. OF PATIENTS /20	PERCENTAGE
1.	Kurinji	3	15%
2.	Mullai	0	0%
3.	Marutham	0	0%
4.	Neithal	17	85%
5.	Paalai	0	0%

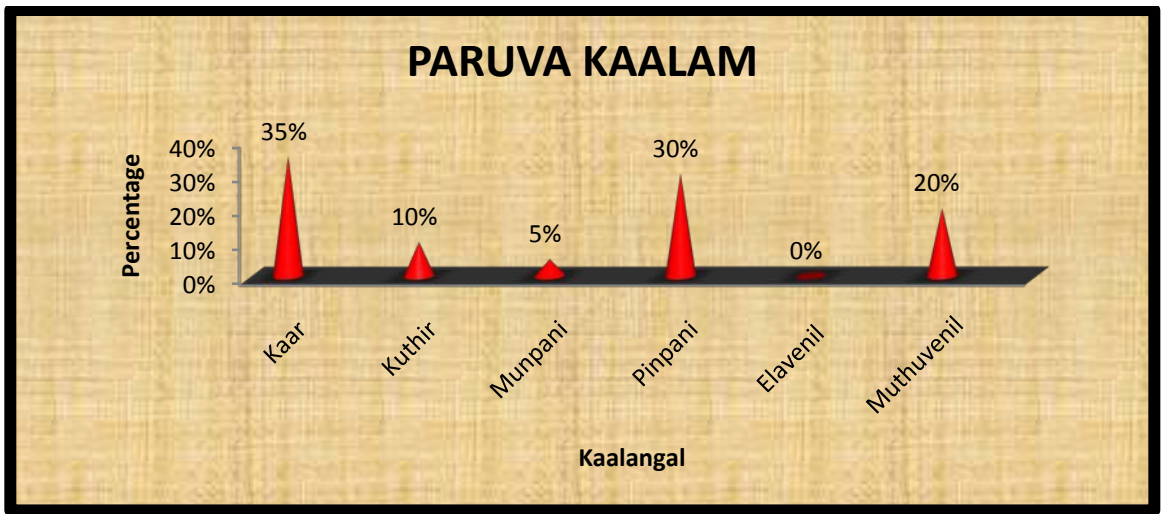


Inference:

From the above data 85% of patient from Neithal and15% of cases from Kurinji.

PARUVAKAALAM:

SL.NO	PARUVAKAALAM	MONTH	NO. OF PATIENTS /20	PERCENTAGE
1.	Kaar Kaalam	Avani, Puratasi, Mid Aug-Mid Oct	7	35%
2.	Kuthir Kaalam	Iyppasi, Kaarthigai Mid Oct-Mid Dec	2	10%
3.	Munpani Kaalam	Margazhi, Thai Mid Dec-Mid Feb	1	5%
4.	Pinpani Kaalam	Maasi, Panguni Mid Feb-Mid April	6	30%
5.	Elavenil Kaalam	Chithirai, vaigasi Mid April- MidJune	0	0%
6.	Muthuvenil Kaalam	Aani, Aadi Mid June-Mid Aug	4	20%

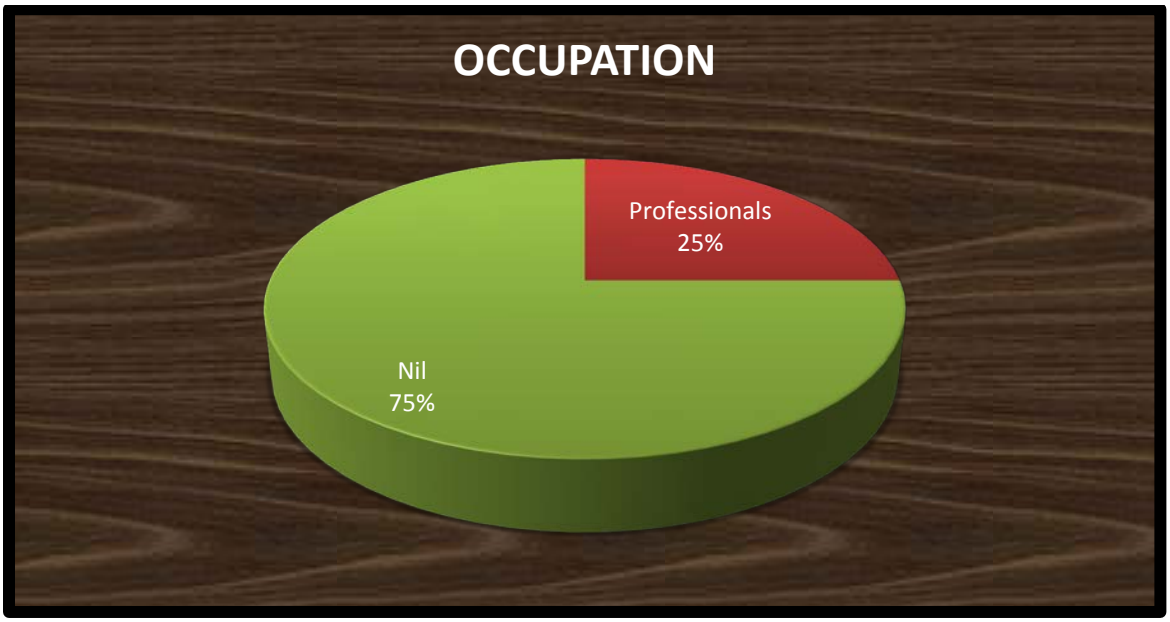


Inference:

35% of case came in Kaar kaalam and 30% of case in Pinpani kaalam, 20% of cases in Muthuvenil kaalam, 10% of cases in Kuthir kaalam and 5% of cases in Munpani kaalam.

OCCUPATIONAL STATUS:

SL.NO	OCCUPATION	NO OF PATIENT /20	PERCENTAGE
1.	Professionals	5	25%
2.	Retired persons	15	75%



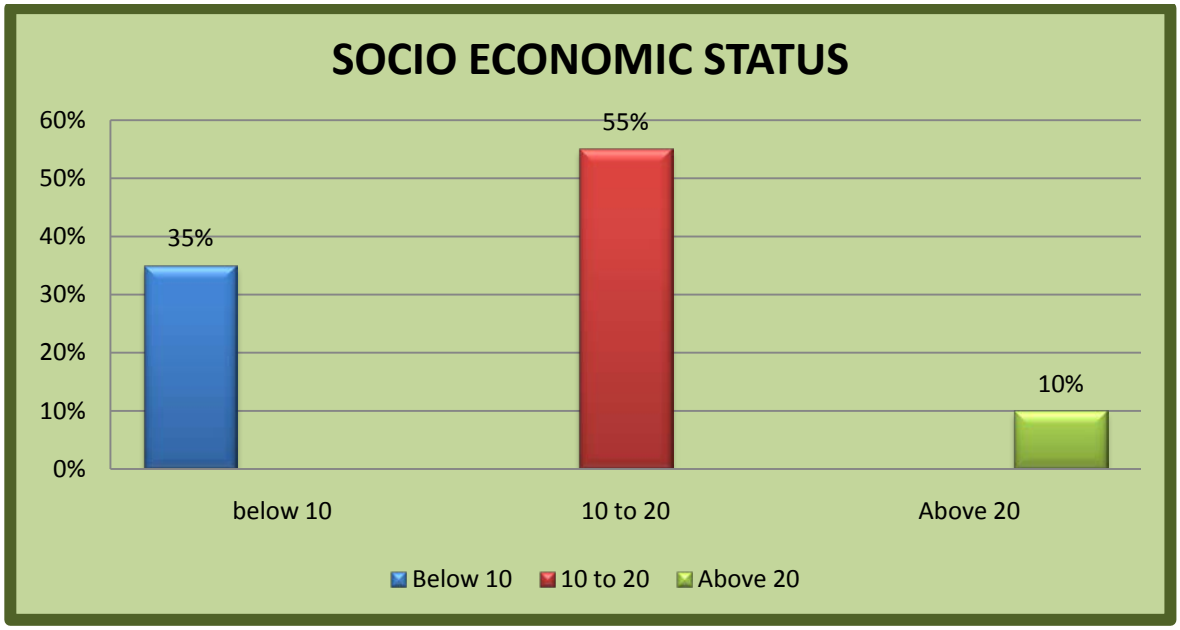
Inference:

25% of cases were Professionals.

75% of cases were Retired persons.

SOCIO ECONOMIC STATUS*:*

Sl.No.	*Socio Economic Status*	No.of Patients /20	Percentage
1.	Low income group (below 10000/month)	7	35%
2.	Middle income group (10000-20000/ month)	11	55%
3.	High income group (above 20000/month)	2	10%

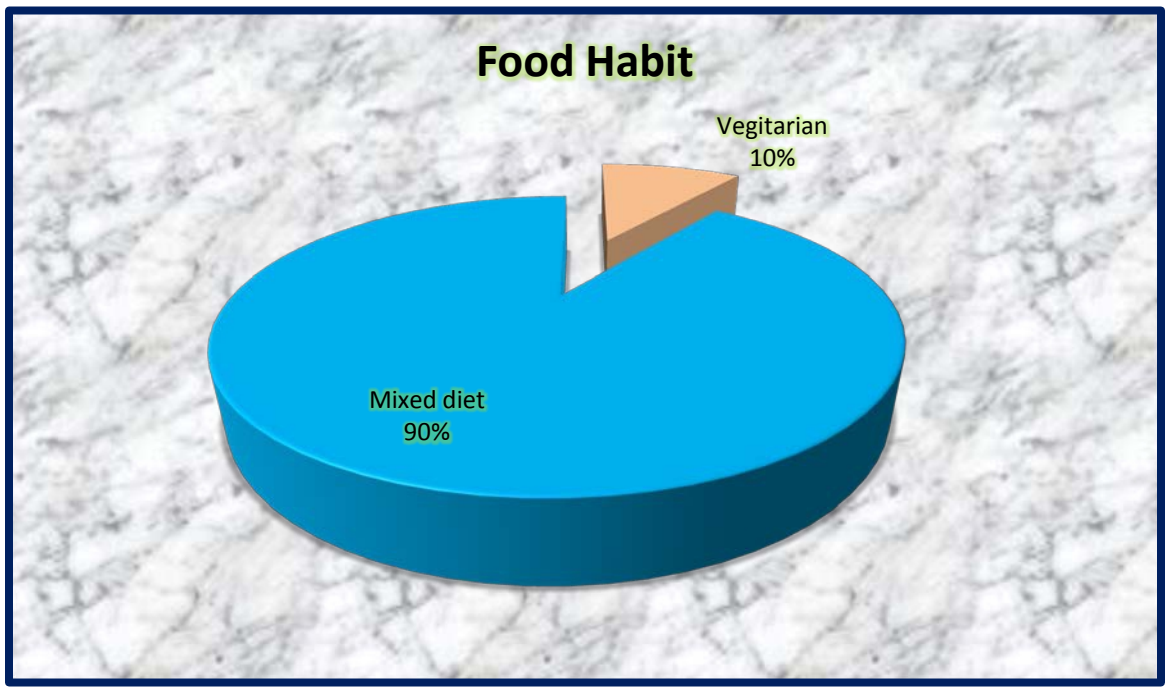


Inference:

55% of cases belong to middle income group and 35% of patients belong to lower income group. 10% of cases belong to high income group.

FOOD HABITS*:*

SL.NO.	FOOD HABIT	NO. OF PATIENT / 20	PERCENTAGE
1.	Vegetarian	2	10%
2.	Mixed diet including Non-vegetarian	18	90%



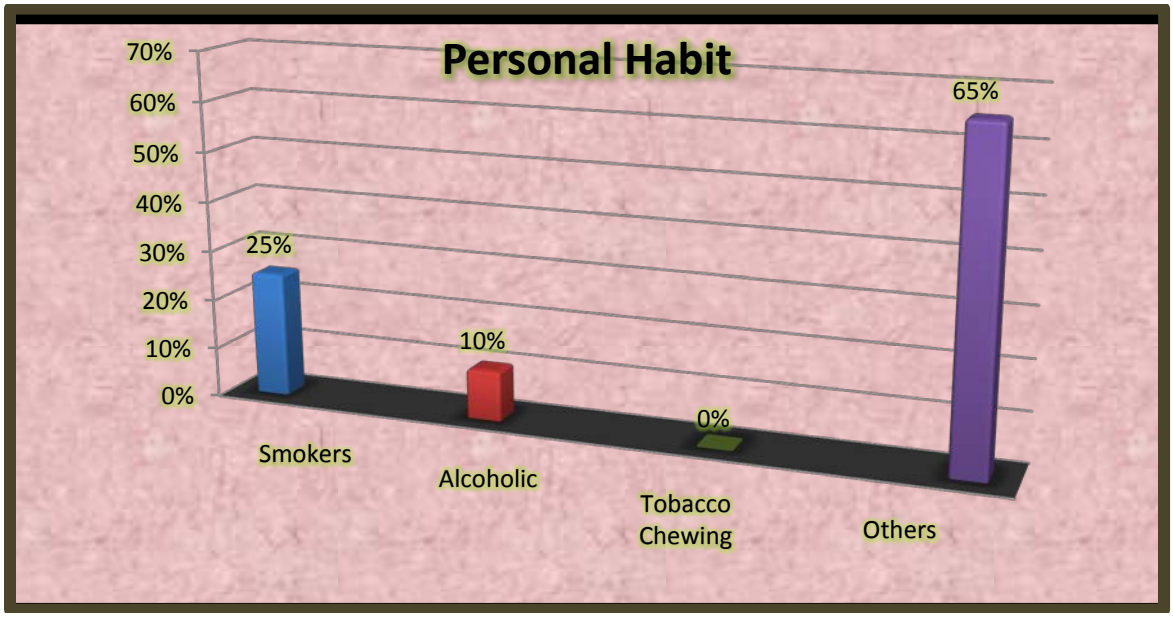
Inference:

90% of cases were mixed diet including Non- vegetarian.

10% of cases were Vegetarian.

PERSONAL HABIT:

SL.NO.	PERSONAL HABIT	NO. OF PATIENT / 20	PERCENTAGE
1.	Smoker	5	25%
2.	Alcoholic	2	10%
3.	Tobacco chewing	0	0%
4.	Others	13	65%

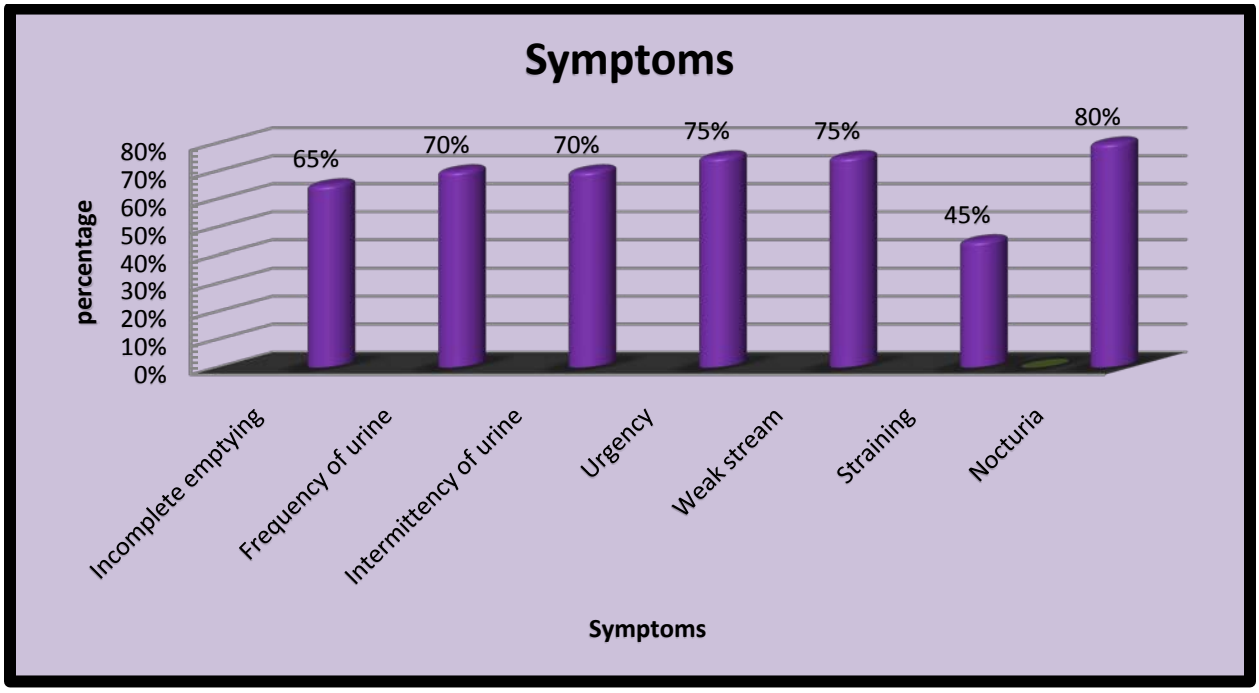


Inference:

65% of patients had no bad habits, 25% of cases were smoker and 10% of cases were alcoholic, 0% of cases were Tobacco Chewing.

SYMPTOMS:

SL.NO	SYMPTOMS	NO. OF PATIENTS/ 20	PERCENTAGE
1.	Incomplete emptying	13	65%
2.	Frequency of urine	14	70%
3.	Intermittency of urine	14	70%
4.	Urgency	15	75%
5.	Weak stream	15	75%
6.	Straining	9	45%
7.	Nocturia	16	80%



Inference:

65% of cases had Incomplete emptying, 70% of cases had Frequency of urine, 70% of cases had Intermittency of urine, 75% of cases had Urgency, 75% of cases had Weak stream, 45% of cases had Straining, 80% of cases had Nocturia.

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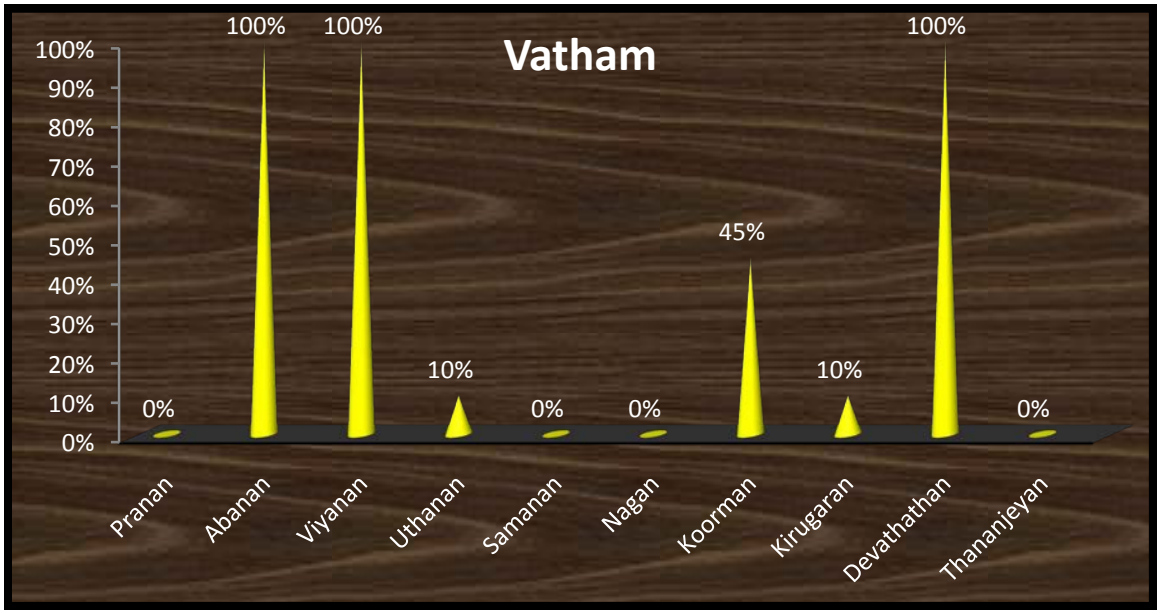
VATHAM:

SL.NO.	VATHAM	NO. OF PATIENT / 20	PERCENTAGE
1.	Pranan	0	0%
2.	Abanan	20	100%
3.	Viyanan	20	100%
4.	Uthanan	2	10%
5.	Samanan	0	0%
6.	Nagan	0	0%
7.	Koorman	9	45%
8.	Kirugaran	2	10%
9.	Devathathan	20	100%
10.	Thananjeyan	0	0%

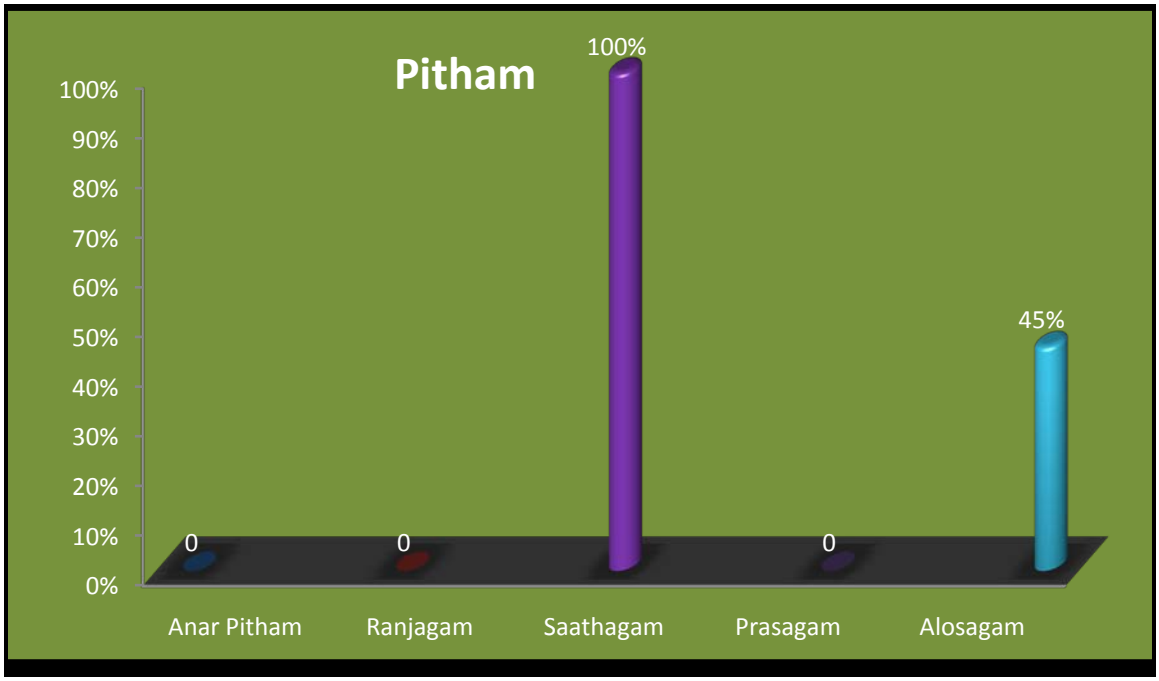


Inference:

Abaanan, viyanan, devathathan were affected in 100% of patients, Koorman was affected in 45% of patients anduthanan and kirugaran were affected in 10% of patients.

PITHAM*:*

SL.NO.	PITHAM	NO.OF PATIENT /20	PERCENTAGE
1.	Anar pitham	0	0%
2.	Ranjagam	0	0%
3.	Saathagam	20	100%
4.	Prasagam	0	0%
5.	Alosagam	9	45%

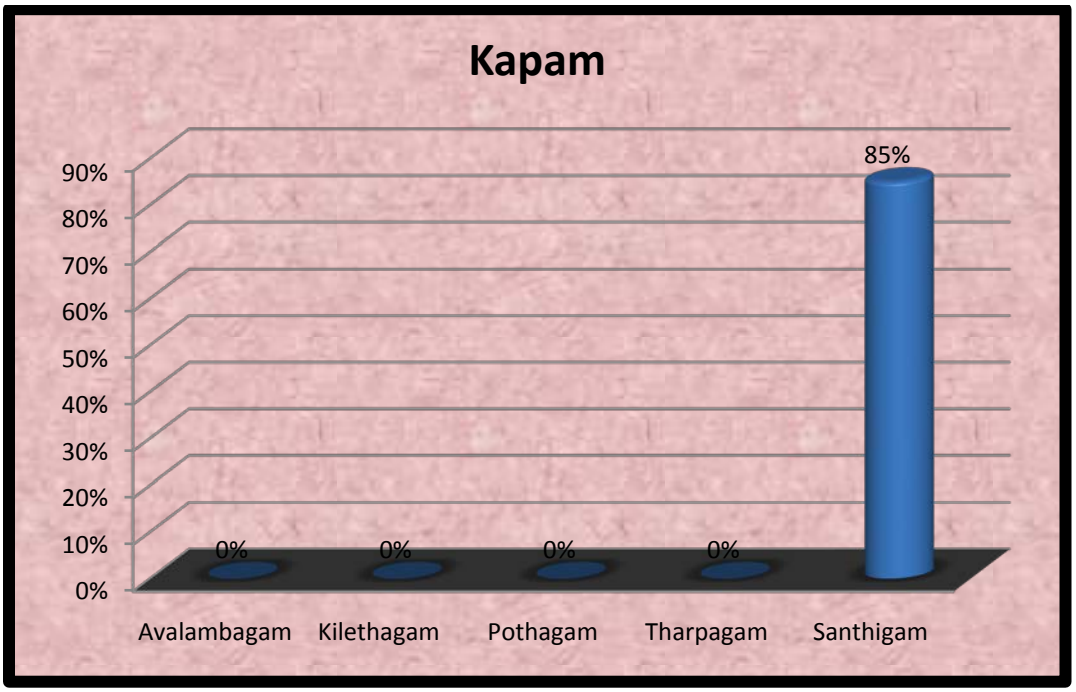


Inference:

Sathagam was affected in 100% of cases, Alosagam was affected in 45% of patients.

KAPAM:

SL.NO.	KAPAM	NO. OF PATEINTS /20	PERCENTAGE
1.	Avalambagam	0	0%
2.	Kilethagam	0	0%
3.	Pothagam	0	0%
4.	Tharpagam	0	0%
5.	Santhigam	17	85%

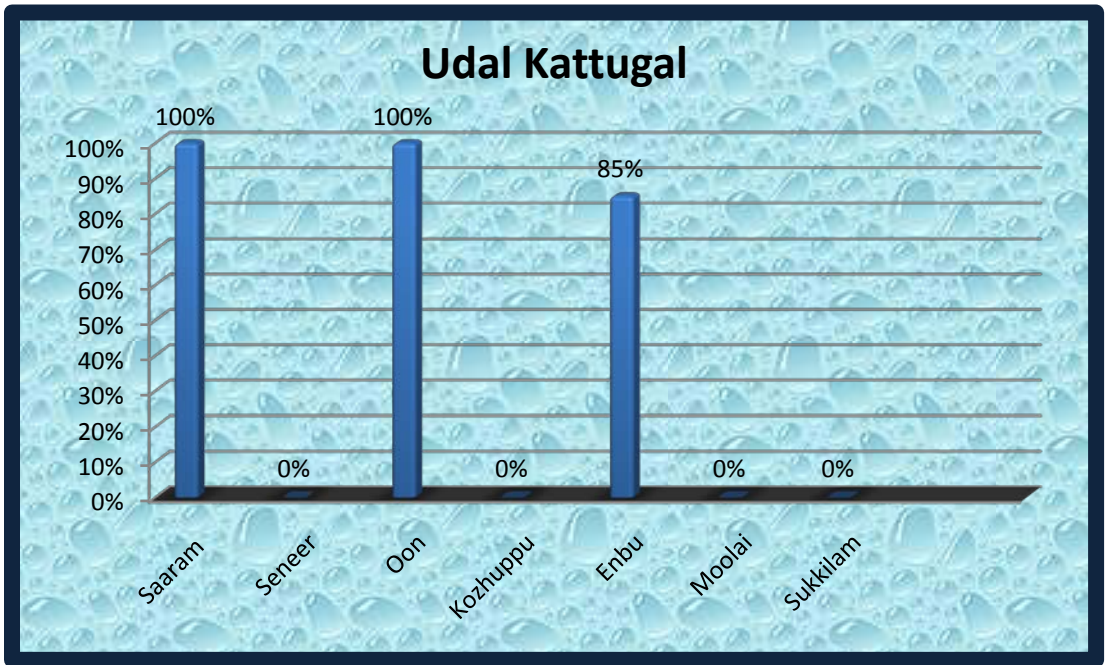


Inference:

Santhigam was affected in 85% of patients.

UDAL KATTUGAL*:*

SL.NO.	UDAL KATTUGAL	NO. OF PATIENT / 20	PERCENTAGE
1.	Saaram	20	100%
2.	Seneer	0	0%
3.	Oon	20	100%
4.	Kozhuppu	0	0%
5.	Enbu	17	85%
6.	Moolai	0	0%
7.	Sukkilam	0	0%

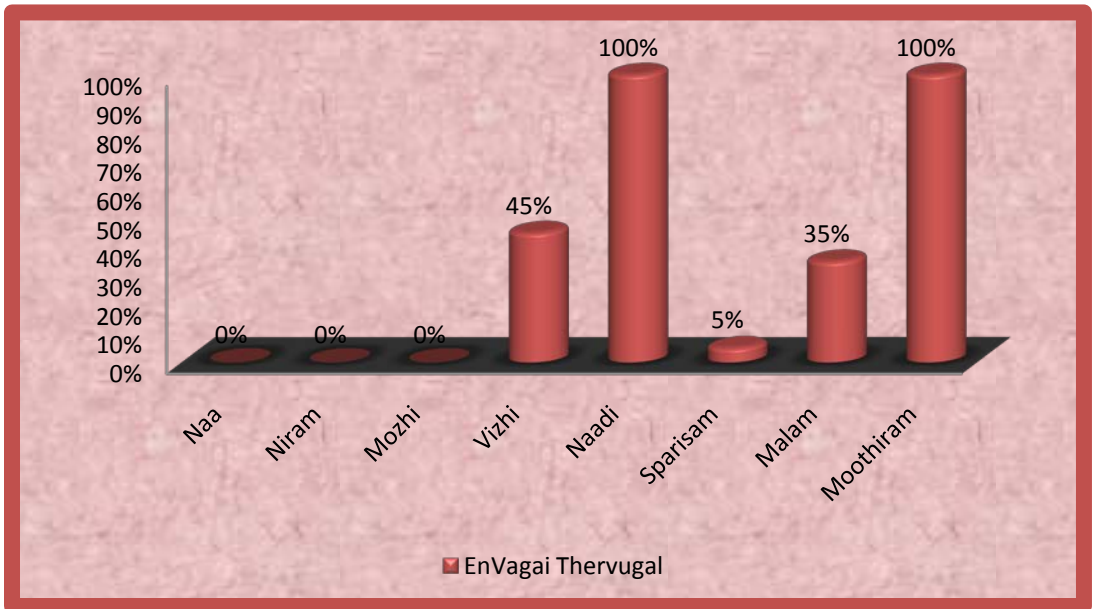


Inference:

Both Saaram and Oon were affected in 100% of patients and Enbu was affected in 85% of patients.

ENVAGAI THERVUGAL:

SL.NO.	ENVAGAI THERVUGAL	NO. OF PATIENT / 20	PERCENTAGE
1.	Naa	0	0%
2.	Niram	0	0%
3.	Mozhi	0	0%
4.	Vizhi	9	45%
5.	Naadi	20	100%
6.	Sparisam	1	5%
7.	Malam	7	35%
8.	Moothiram	20	100%

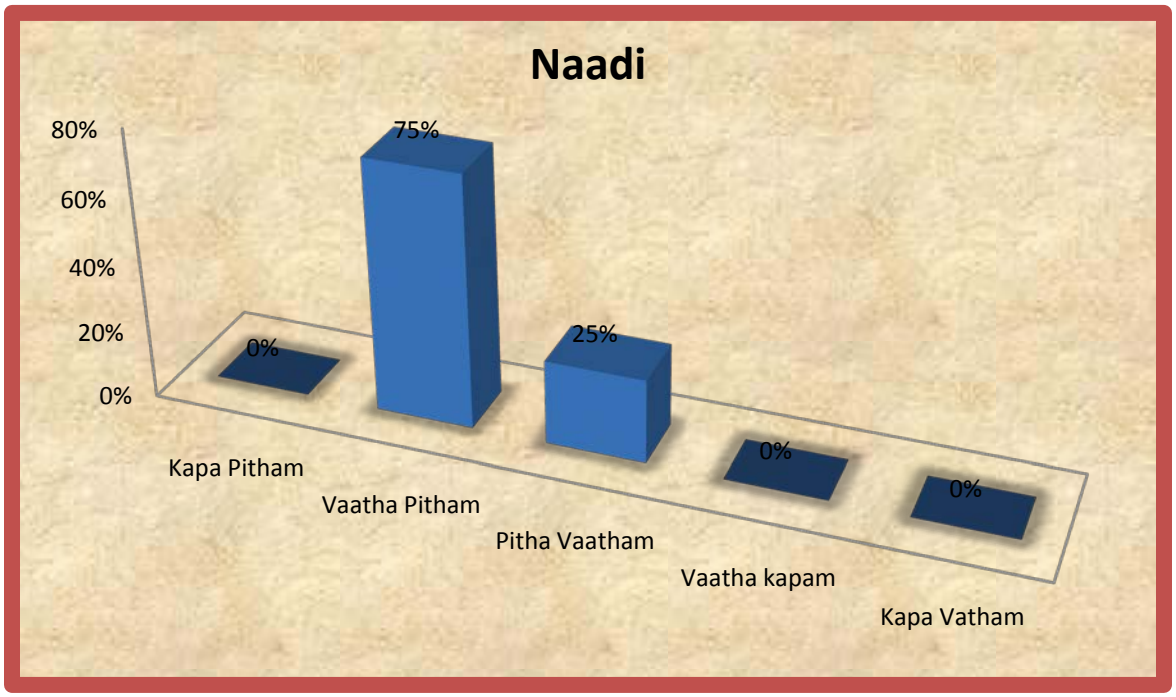


Inference:

Naadi and moothiram were affected in 100% of patients and 45% of patients vizhi was affected, 35% of patients Malam was affected, 5% of patients sparisam was affected.

NAADI:

SL.NO.	NAADI	NO. OF PATIENT / 20	PERCENTAGE
1.	Kappa Pitham	0	0%
2.	Vaatha Pitham	15	75%
3.	Pitha Vaatham	5	25%
4.	Vaatha Kapam	0	0%
5.	Kapa Vaatham	0	0%

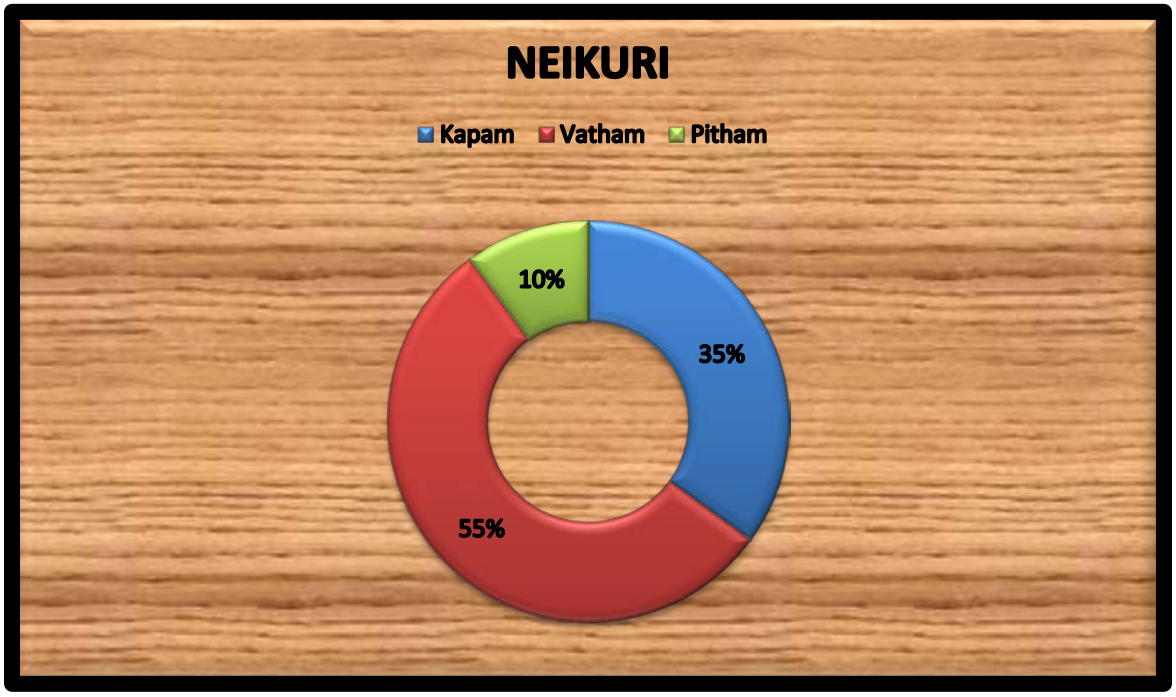


Inference:

75% of patient's vatha pitham naadi was felt and 25% of cases pitha vatha naadi was felt.

NEIKURI:

SL.NO.	NEIKURI	NO. OF PATIENT / 20	PERCENTAGE
1.	Vatham (Spreads like Snake)	11	55%
2.	Pitham (Spreads like Ring)	2	10%
3.	Kapam (Stands like Pearl)	7	35%

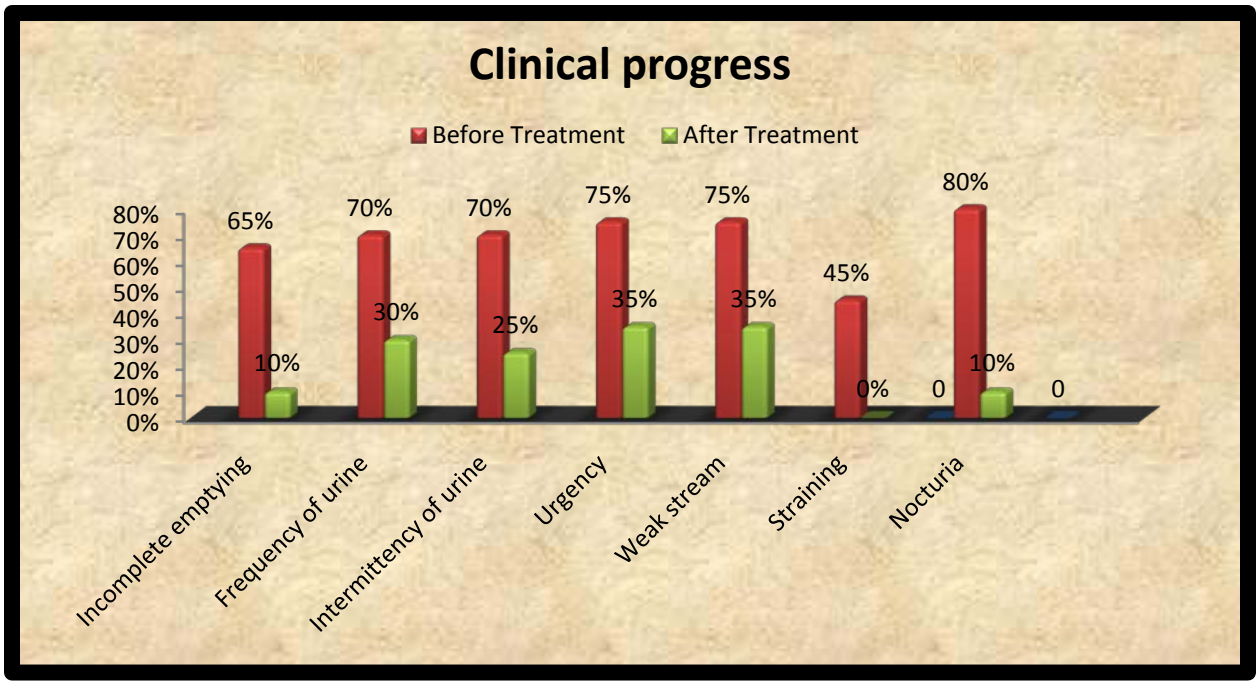


Inference:

55% of cases show Vatha neikuri, 35% shows Kapha neikuri and 10% shows Azhal neikuri.

CLINICAL PROGRESS:

SL.NO	SYMPTOMS	NO. OF PATIENTS/ 20		PERCENTAGE	
		Before Treatment	After Treatment	Before Treatment	After Treatment
1.	Incomplete emptying	13	2	65%	10%
2.	Frequency of urine	14	6	70%	30%
3.	Intermittency of urine	14	5	70%	25%
4.	Urgency	15	7	75%	35%
5.	Weak stream	15	7	75%	35%
6.	Straining	9	0	45%	0%
7.	Nocturia	16	2	80%	10%

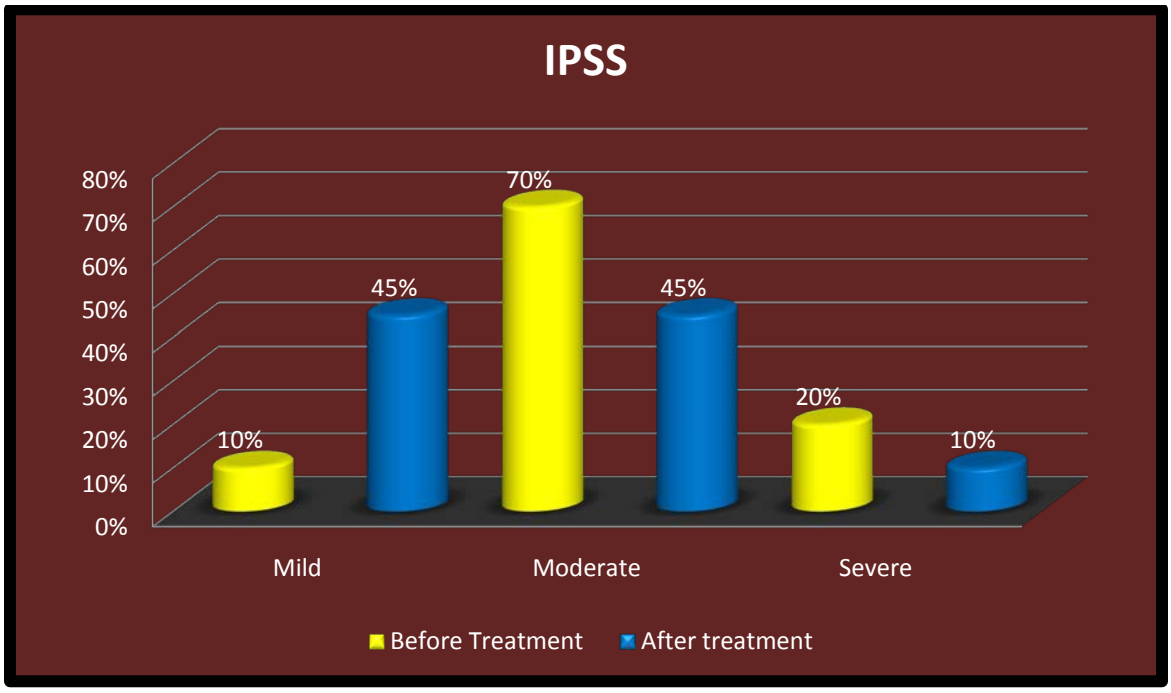


Inference

Before treatment 65% of cases had Incomplete emptying, 70% of cases had Frequency of urine, 70% of cases had Intermittency of urine, 75% of cases had Urgency, 75% of cases had Weak stream, 45% of cases had Straining, 80% of cases had Nocturia. After treatment Straining were completely relieved and Nocturia and Incomplete emptying 10% and 25% of cases Intermittency of urine.

IPSS PROGRESS:

SL.NO	IPSS	NO OF PATIENTS / 20		PERCENAGE	
		Before treatment	After treatment	Before treatment	After treatment
1.	Mild (0- 7)	2	9	10%	45%
2.	Moderate (8 – 19)	14	9	70%	45%
3.	Severe (20 – 35)	4	2	20%	10%

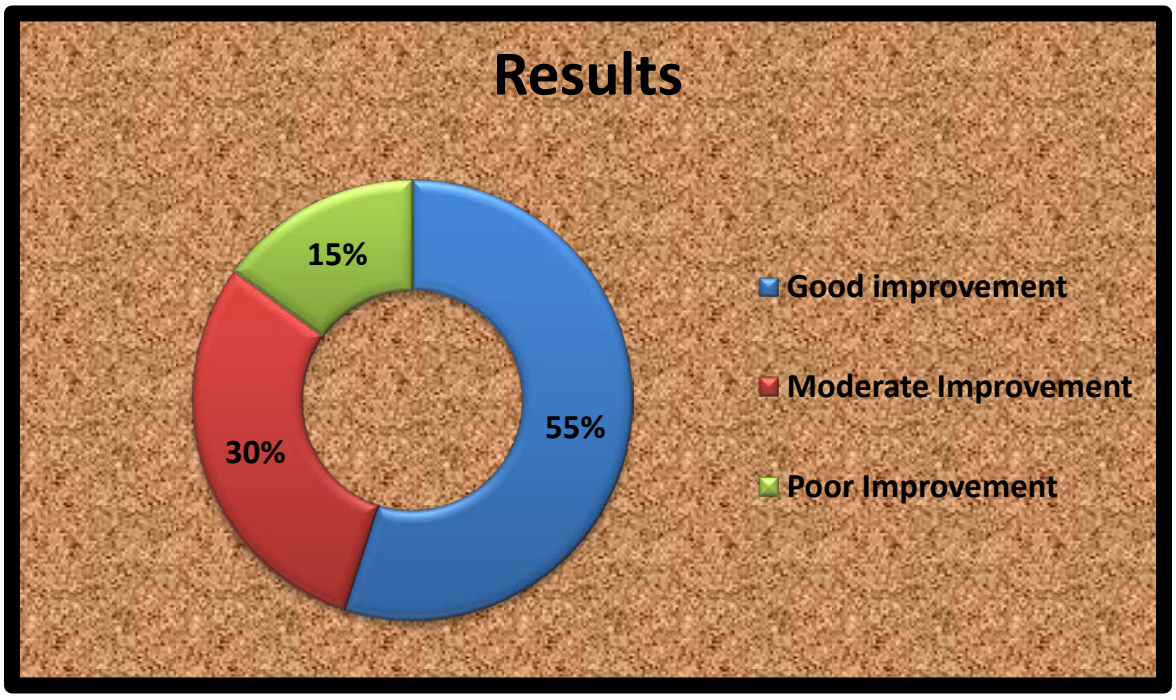


Inference:

Before treatment 10% of cases had Mild symptoms, 70% of cases had Moderate symptoms, 20% of cases had severe symptoms. After treatment,45% of cases come to Mild Symptom category 45% of cases come under moderate category, and 10% of cases come under severe category.

GRADATION OF RESULTS:

SL.NO.	Results	NO. OF PATIENT / 20	PERCENTAGE
1.	Good improvement	11	55%
2.	Moderate Improvement	6	30%
3.	Poor Improvement	3	15%



Inference:

55% of Patients show good improvement, 30% of shows moderate improvement and 15% of cases shows poor improvement.

LABORATORY INVESTIGATION REPORT (OP)

SL. NO.	OP. NO.	NAME	AGE	HEAMOTOLOGICAL REPORT														URINE ANALYSIS						STOOL EXAMINATION			
				BEFORE TREATMENT				AFTER TREATMENT				ESR(mm)				HB(Gm)		BT			AT			BT		AT	
				TC (Cu/mm)	DC			TC (Cu/mm)	DC			BT		AT		BT	AT										
					P	L	E		P	L	E	½ Hr	1 Hr	½ Hr	1 Hr			Alb	Sug	Dep	Alb	Sug	Dep	Ova	Cyst	Ova	Cyst
1.	3619	Mr.Sheshatri	56	11,000	55	42	3	10,600	60	36	3	15	25	10	13	13.2	13	Nil	Nil	OEC	Nil	Nil	Nil	Nil	Nil	Nil	Nil
2.	7740	Mr.Gangatharan	64	9,800	56	38	6	9,800	60	34	8	15	30	12	18	15	14.5	Nil	Nil	OEC	Nil	Nil	OPC	Nil	Nil	Nil	Nil
3.	8494	Mr.Balakrishnan	72	10,400	58	39	3	11,000	58	40	4	5	22	4	10	12.2	12.5	Nil	Nil	OEC	Nil	Nil	OPC	Nil	Nil	Nil	Nil
4.	2179	Mr.Nadesan	74	11,000	59	37	4	10,500	55	42	5	5	10	8	16	12.4	12.5	Nil	Nil	FEC	Nil	Nil	Nil	Nil	Nil	Nil	Nil
5.	636	Mr.Thiruvengadam	68	9,700	58	36	6	9,800	56	40	2	3	5	4	8	12	12	Nil	Nil	FEC	Nil	Nil	Nil	Nil	Nil	Nil	Nil
6.	3155	Mr.Rangarajan	61	11,300	55	35	6	11,400	60	35	3	7	12	10	14	13	12.5	Nil	Nil	OPC	Nil	Nil	Nil	Nil	Nil	Nil	Nil
7.	5269	Mr.Ramasamy	75	11,600	56	35	4	10,100	62	34	4	3	5	4	10	13	12.8	Nil	Nil	FPC	Nil	Nil	Nil	Nil	Nil	Nil	Nil
8.	9954	Mr.Ganesan	58	10,100	62	34	4	10,000	62	28	4	11	32	6	10	12	13	Nil	Nil	FEC	Nil	Nil	OPC	Nil	Nil	Nil	Nil
9.	2129	Mr.Kannan	62	9,600	59	35	6	9,500	58	30	3	3	7	3	6	15	15	Nil	Nil	FEC	Nil	Nil	Nil	Nil	Nil	Nil	Nil
10.	5782	Mr.Gopikrishnan	54	9,900	60	37	4	9,800	55	35	6	10	20	9	18	13.8	13.7	Nil	Nil	OPC	Nil	Nil	Nil	Nil	Nil	Nil	Nil
11.	4592	Mr.Sanmugam	75	9,700	55	32	5	10,000	56	35	4	2	4	3	6	14.8	15	Nil	Nil	FPC	Nil	Nil	Nil	Nil	Nil	Nil	Nil
12.	1647	Mr.Jeganathan	55	9,800	57	28	3	10,100	58	34	5	6	10	5	12	14.6	14.6	Nil	Nil	FPC	Nil	Nil	Nil	Nil	Nil	Nil	Nil
13.	695	Mr.Govindaraj	62	9,700	58	35	7	9,800	65	40	6	11	20	7	12	11	12	Nil	Nil	OEC	Nil	Nil	Nil	Nil	Nil	Nil	Nil
14.	1799	Mr.Jeyaraman	75	11000	58	37	2	10,400	55	32	5	2	5	4	10	13	12.8	Nil	Nil	FEC	Nil	Nil	Nil	Nil	Nil	Nil	Nil
15.	2193	Mr.Francis	68	10,800	66	30	4	10,700	55	36	3	2	5	3	6	14	14.2	Nil	Nil	OPC	Nil	Nil	OPC	Nil	Nil	Nil	Nil
16.	189	Mr.Ramachandran	77	9700	57	38	5	10,700	60	37	4	3	5	3	8	12.8	13	Nil	Nil	FPC	Nil	Nil	Nil	Nil	Nil	Nil	Nil
17.	9445	Mr.Krishnan	65	11,800	58	38	4	9,800	55	32	5	2	5	4	10	13	12.8	Nil	Nil	Nil	Nil	Nil	OPC	Nil	Nil	Nil	Nil
18.	716	Mr.Kumaresan	57	10,800	60	32	8	9,600	60	38	2	10	22	9	18	12.4	12.5	Nil	Nil	FEC	Nil	Nil	OPC	Nil	Nil	Nil	Nil
19.	7052	Mr.Nadarajan	60	10,100	54	42	4	11,200	55	30	4	7	17	8	18	14	14.1	Nil	Nil	OPC	Nil	Nil	OPC	Nil	Nil	Nil	Nil
20.	6412	Mr.Guberan	68	9,900	59	37	4	10,800	60	35	6	12	24	10	15	13	13	Nil	Nil	FEC	Nil	Nil	FPC	Nil	Nil	Nil	Nil

TC – Total Count
Dc – Differential Count
P – Polymorph
L – Lymphocyte
E – Eosinophil

Hb – Haemoglobin
ESR – Erythrocyte Sedimentation Rate
Alb – Albumin
Sug – Sugar
Dep – Deposits

OEC – Occasional Epithelial Cells
OPC – Occasional Pus Cells
FPC – Few Pus Cells
FEC – Few Epithelial Cells

NO. OF PATIENTS – BEFORE TREATMENT AND AFTER TREATMENT

SL.NO.	OP. NO	NAME	AGE	SCREENING TEST (Serum total PSA ng/ml)	USG PELVIS - PROSTATE						NO. OF DAYS	RESULT
					BEFORE TREATMENT			AFTER TREATMENT				
					Measurement (cm)	Volume (cc)	PRU (ml)	Measurement (cm)	Volume (cc)	PRU(ml)		
1.	3619	Mr.Sheshatri	56	**0.50**	3.8*3.2*3.1	20	43	**3.8*3.0*3**	**20**	**18**	**30**	Good
2.	7740	Mr.Gangatharan	64	1.85	5.2*5.8*5.2	84	38	5.6*5.1*4.9	78	80	45	Good
3.	8494	Mr.Balakrishnan	72	0.72	4.5*4.6*3.2	30	54	4.5*5.0*3	30	50	45	Poor
4.	2179	Mr.Nadesan	74	2.03	3.6*4.6*4.7	45	60	3.5*4.2*4.0	45	51	30	Moderate
5.	636	Mr.Thiruvengadam	68	1.85	3.8*4.2*3.7	29	28	3.2*4*3.1	27	9	30	Good
6.	3155	Mr.Rangarajan	61	0.23	4.9*3.7*3.4	32	15	4.8*3.5*4.0	30	7	30	Moderate
7.	5269	Mr.Ramasamy	75	0.95	4.6*3.8*3.3	30	40	4.7*3.2*3	28	35	45	Moderate
8.	9954	Mr.Ganesan	58	3.00	3.8*4.2*3.7	45	100	3.9*4*3.1	38	95	30	Moderate
9.	2129	Mr.Kannan	62	0.925	4.3*3.5*2.9	23	96	4.3*4.1*2.5	25	100	45	Poor
10.	5782	Mr.Gopikrishnan	54	1.45	5*3.5*3	28.5	164	4.1*3.2*2.8	25	125	45	Good
11.	4592	Mr.Sanmugam	75	2.52	4.3*4.3*3.5	33.7	85	4.1*4.0*3.2	20	72	45	Good
12.	1647	Mr.Jeganathan	55	0.69	4.2*3.6*3.5	28	96	4.0*3.6*3.2	15	25	30	Good
13.	695	Mr.Govindaraj	62	2.56	4.5*5.1*4.7	56.7	250	4.2*4.0*3.8	34	40	45	Good
14.	1799	Mr.Jeyaraman	75	0.90	4.4*4.5*4.6	47	100	4.4*4.5*3.8	23.5	110	30	Moderate
15.	2193	Mr.Francis	68	0.82	5.6*7.2*5.8	45	150	5.5*6.9*5	41	75	45	Good
16.	189	Mr.Ramachandran	77	2.30	4.5*4.2*3.2	32.95	94	4.5*3.3*2.8	30	70	45	Good
17.	9445	Mr.Krishnan	65	1.53	3.6*4.6*4.6	42	6	3.1*4.1*4.6	40	8	45	Moderate
18.	716	Mr.Kumaresan	57	3.02	5.7*3.6*4.4	47	140	5.1*4.2*4.3	35.5	80	30	Good
19.	7052	Mr.Nadarajan	60	0.86	4.8*3.7*4.3	32	65	4.9*4.1*5	35	118	45	Poor
20.	6412	Mr.Guberan	68	2.98	4.9*4.4*4.1	47	120	4.2*4.0*3.8	33.7	92	30	Good

PRU – Post Void Residual Urine

NO. OF PATIENTS – BEFORE TREATMENT AND AFTER TREATMENT

SL.NO.	OP. NO	NAME	AGE	IPSS (TOTAL)		Result
				Before Treatment	After Treatment	
1.	3619	Mr.Sheshatri	56	7	5	Good
2.	7740	Mr.Gangatharan	64	19	12	Good
3.	8494	Mr.Balakrishnan	72	32	28	Poor
4.	2179	Mr.Nadesan	74	18	10	Moderate
5.	636	Mr.Thiruvengadam	68	17	7	Good
6.	3155	Mr.Rangarajan	61	17	12	Moderate
7.	5269	Mr.Ramasamy	75	15	7	Moderate
8.	9954	Mr.Ganesan	58	11	6	Moderate
9.	2129	Mr.Kannan	62	19	13	Poor
10.	5782	Mr.Gopikrishnan	54	14	7	Good
11.	4592	Mr.Sanmugam	75	13	6	Good
12.	1647	Mr.Jeganathan	55	7	4	Good
13.	695	Mr.Govindaraj	62	28	12	Good
14.	1799	Mr.Jeyaraman	75	24	16	Moderate
15.	2193	Mr.Francis	68	16	9	Good
16.	189	Mr.Ramachandran	77	16	8	Good
17.	9445	Mr.Krishnan	65	13	8	Moderate
18.	716	Mr.Kumaresan	57	10	5	Good
19.	7052	Mr.Nadarajan	60	27	24	Poor
20.	6412	Mr.Guberan	68	14	7	Good

IPSS – International Prostate Symptoms Score

0-7 : Mildly Symptomatic

8-19 : Moderately Symptomatic

20-35 : Severely Symptomatic

Vijaya Diagnostic and Research Centre

VIJAYA HOSPITAL
434 (Old # 180), N.S.K. SALAI
CHENNAI - 600 026
Phone: 24802221
24802165
24802701
Extn. 273

Name : MR. G. GOVINDA RAJ
Age/Sex : 59/MALE
Refered By : DR.CC
VisitDate : 5/04/12

ULTRASOUND STUDY FOR KUB REGION

RIGHT KIDNEY :
Measures 9.8 x 4.1 cms
Shows normal size, shape & position.
No dilatation of pelvicalyceal system is seen.
Cortico medullary differentiation maintained.
No calculi.

LEFT KIDNEY :
Measures 9.2 x 4.0 cms
Shows normal size, shape & position.
No dilatation of pelvicalyceal system is seen.
Cortico medullary differentiation maintained.
No calculi.

URINARY BLADDER :
Well distended with circumferential wall thickness with irregularities seen.
No evidence of any calculus.

PROSTATE :
Measures 4.5 x 5.1 x 4.7 cms
Weight 56.7 gms
Intravesical extension measuring 1.0 cm noted.
Shows homogenous echoes.
Seminal vesicles appear normal.

POST VOID :
Measures 250 cc

IMPRESSION :
-BLADDER OUTLET OBSTRUCTION.
-PROSTATOMEGALY.
-SIGNIFICANT RESIDUAL URINE.

DR.SIVA SUNDAR, D.M.R.D, D.N.B.
(Radiologist)

This imaging film(s) and reports are not meant for medico-legal purposes.
Please correlate with clinical findings and other biochemical and pathological reports.

GEMINI
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822, Chennai Tiruvallur High Road
Ambattur, Chennai - 600 052
Phone : 044-2658 4417, 2658 4418
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Quality You Can Depend On . . . .

CONSULTANT RADIOLOGISTS

Dr. G. Godwin, M.D., DNB | Dr. V. Ramkumar, DMRD, DNB | Dr. R. Chitrah, MD | Dr. V. Senthil, DMRD, DNB

Name : Mr.Govindaraj
Age : 60 Y/M
Ref.By.: Dr.K.Samraj.,

Date : 03.08.2012
Id.No.: Ak12/6346

Ultrasound - KUB

Kidneys:

RT.Kidney measures 9.5 x 5.1cms.

Calculus measuring 3-4mms is visualized in lower pole calyx of right kidney.

LT.Kidney measures 9.2 x 5.0cms.

Cortico medullary differentiation is maintained on both sides.

Pelvicalyceal system and the ureters on both sides appear normal.

Bladder:

Is normal in contour. No intra luminal echoes are seen.

Mild diffuse urinary bladder wall thickening visualized.

Post-void residual urine volume – 40ml.

Prostate:

Enlarged in size and measures 4.2 x 4.0 x 3.8cms. Volume 34cc.

Nodular projection of the enlarged prostate into the urinary bladder noted.

No focal lesion is seen.

Impression:

- **Right renal calculus.**
- **Mild diffuse urinary bladder wall thickening visualized.**
- **Prostatomegaly with post-void residual urine volume – 40ml.**

Dr.V.Sivakumar.,MD.,
Radiologist

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Advanced MRI & CT Scan

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Quality You Can Depend On ****

CONSULTANT RADIOLOGISTS

Dr. G. Godwin, M.D., DNB | Dr. V. Ramkumar, DMRD, DNB | Dr. R. Chitrah, MD | Dr. V. Senthil, DMRD, DNB

Name : Mr.Gangatharan
Age : 64 Y/M
Ref.By.: Dr.K.Samraj.

Date : 30.01.2012
Id.No.: Usg - 13577

Ultrasound KUB

Kidneys:

RT.Kidney measures 9.0 x 4.3cms.

LT.Kidney measures 9.5 x 4.7cms.

Cortico medullary differentiation is maintained on both sides.

Pelvicalyceal system and the ureters on both sides appear normal.

No evidence of calculus is seen.

Bladder:

Is normal in contour.
Diffuse thickening of the urinary bladder wall measuring 5.2mms is visualized.
A focal polypoidal mass measuring 1.1 x 1.1cms is visualized in left posterolateral wall of urinary bladder.
Minimal vascularity is seen within the mass.
Post-void residual urine volume - 38ml.

Prostate:

Enlarged in size and measures 5.2 x 5.8 x 5.2cms. Volume 84ml.

Impression:

- **Focal polypoidal mass in left posterolateral wall of urinary bladder -Suggested cystoscopic biopsy for further evaluation.**
- **Prostatomegaly with post void residual urine volume of 38ml.**
- **Diffuse thickening of the urinary bladder wall visualized - ? Cystitis / chronic bladder outlet obstruction.**

Dr.R.Chitrah Sivan.,MD.,
Consultant Radiologist.
Ph.No:98848 15192.

Kidneys - (N).

Ubladder - (N)

Prostate - Enlarged in size. It measures ~56 x 57 x 49mm (Vol ~73 ml). Transverse enlargement of prostate noted.

Significant post void residual urine in bladder (Vol ~88 ml).

Impn - prostatomegaly c significant post void residual urine in bladder (Vol ~88 ml)

- for PSA correlation.

Discussion

DISCUSSION

Benign prostatic hyperplasia is the common geriatric disease in the world wide. Even though the BPH is not a major disease in elderly people. When it occurring at old age it becomes a severe illness.BPH event is increased over the age of 50 and it produces the lower urinary tract obstruction which leads to following symptoms are, Incomplete emptying, Frequency of urine, Intermittency of urine, Urgency, Weak stream, Straining, Nocturia.

Before this illness starts or aggravates the awareness about the BPH is important. So as to prevent the severity, and avoid these symptoms which leads to carcinoma of prostate.

20 patients were treated in outpatient department of Post graduate pothumaruthuvam, Govt Siddha Medical College Hospital, Chennai – 106.

All patients were subjected to preliminary investigations which include haematological, urine examination, USG pelvis are noted before and after Treatment

The Trial Medicine Velvanga parpam, dose 65mg was administered twice daily for 30 days.

Age Distribution*:*

According to the above mentioned data 40% of patients were in age groups 61-70 years, 30% of patients were in age group 71-80 year, 30% of patients were in age group 51-60 years.

.

Distribution of Thinai:

From the above data 85% of patient from Neithal and15% of cases from Kurinji.

Paruvakalam:

According to this study 35% of case came in Kaar kaalam and 30% of case in Pinpani kaalam, 20% of cases in Muthuvenil kaalam, 10% of cases in Kuthir kaalam and 5% of cases in Munpani kaalam.

Occupational Status:

In my study ,25% of cases were Professionals, 75% of cases were Retired persons.

Socio Economic Status*:*

According to this study, 55% of cases belong to middle income group and 35% of patients belong to lower income group. 10% of cases belong to high income group.

Food Habits:

According to this study , 90% of cases were mixed diet including Non-vegetarian, 10% of cases were Vegetarian.

Personal Habits:

In my study 65% of patients had no bad habits, 25% of cases were smoker and 10% of cases were alcoholic, 0% of cases were Tobacco Chewing..

Symptoms:

According to this study 65% of cases had Incomplete emptying, 70% of cases had Frequency of urine, 70% of cases had Intermittency of urine, 75% of cases had Urgency,75% of cases had Weak stream, 45% of cases had Straining, 80% of cases had Nocturia.

Classification of Results According To Vali, Azhal, Iyyam

Vali :

Incomplete emptying, Frequency of urine, Intermittency of urine, Urgency, Weak stream, Straining, Nocturia are due to deranged Abana Vayu. And Abaanan was affected in 100% of patients, Koorman was affected in 45% of patients, Kirugaran was affected 10% of patients and Viyanan, Devathathan was affected in 100% of patients.

Azhal:

Satham was affected in100% of patients, Alosagam was affected in 45% of patients.

Iyyam:

Santhigam was affected in 85% of patients.

Udhal Kattugal:

Both Saaram and Oon were affected in 100% of patients and Enbu was affected in 85% of patients

Envagai Thervu:

Naadi and Moothiram were affected in 100% of patients and 45% of patients vizhi was affected, 35% of patients Malam was affected, 5% of patients sparisam was affected.

Naadi:

75% of patient’s vatha pitham naadi was felt and 25% of cases pitha vatha naadi was felt..

Neikuri

55% of cases show Vatha neikuri, 35% shows Kapha neikuri and 10% shows Azhal neikuri.

Clinical Progress:

Before treatment 65% of cases had incomplete emptying, 70% of cases had Frequency of urine, 70% of cases had Intermittency of urine, 75% of cases had Urgency, 75% of cases had Weak stream, 45% of cases had Straining, 80% of cases had Nocturia. After treatment Straining were completely relieved and Nocturia and Incomplete emptying 10% and 25% of cases Intermittency of urine.

IPSS progress:

Before treatment 10% of cases had Mild symptoms, 70% of cases had Moderate symptoms, 20% of cases had severe symptoms. After treatment,45% of cases come to Mild Symptom category 45% of cases come under moderate category, and 10% of cases come under severe category.

Trial Medicine:

All the 20 patients treated with the Trial Medicine Velvanga parpam with Butter for 15 to 30 days The disease and treatment are based primarily on the derangement of Mukkutram, which again is based on the Pancha bootham theory.

Mukkutra theory:

Ukkara soolai is caused by the derangement of Vatha kutram. That is,

"நெடுவாத சார்பதுவுமின்றி சூலை வராது"

And the trial drug Velvanga parpam has follows,

S. No	Content	Suvai	Bootha serkai
1.	Velvangam	Kaippu	Vali + Vin
2.	Alove vera	Siru kaippu	Vali + Vin

The predominant Bootham present in Velvanga parpam is Vali (vatham).

Predominant suvai present in Velvanga parpam is KAIPPU, And predominant bootham is Vali (Katru).Hence all the content of trial drug has vali in the pancha bootha theory. So being the vatha disease, all the drugs having the vatha(vali) pootha treat the disease in OPPURAI MARUTHUVAM.

Bio chemical analysis:

The result of Bio-chemical analysis reveals that Velvanga parpam contains Acid radicals such as a Chloride, Zinc, Calcium and Magnesium.

Pharmacological study:

Pharmacological study reveals that Velvanga parpam contains Anti-tumour activity The antitumor effect of the drug is evident from the increase in lifespan, reduction in solid tumour volume and also the reversal of altered haematological parameter almost equal to normal .The drug can be used as a novel potential agent in the area of tumour chemotherapy.

Bio-statistical report:

The Bio-statistical report reveals that the result of the treatment shows significant result P value <0.01 , Before and After treatment Mean value for IPSS (International Prostate Symptoms Score) -6.55±3.02.

The above outcomes obtained from the clinical study were better and much encouraging.

Summary

SUMMARY

The aim of the study is to reduce the symptoms of Ukkara soolai patients. The trial medicine Velvanga parpam was prepared as per literature. The duration of the trial period is 30 days. The trial dose is Velvanga parpam 65 mg twice daily with butter. I had selected 20 patients for the trial based on Inclusion and Exclusion criteria.

Before treatment routine blood, urine, PSA and USG-Pelvis taken in all 20 patients. Siddha parameters like udal thathukkal, Envagai thervu, Neerkuri and Neikuri were noted in case sheet proforma. Patients were instructed to come for next review once in 7 days.

Patients were come with clinical symptoms like Incomplete emptying, Frequency of urine, Intermittency of urine, Urgency, Weak stream, Straining, Nocturia. The entire details of the patients were noted in the case sheet proforma.

Age :

Most of the patients were in the age group between 61-70 years.

Thinai :

Most of the patients were from Neithal Thinai 85%.

Kalam :

Most of the patients were affecting in Karkaalam and pinpanikaalam.

Diet & Personal habits:

People with habit of taking Vegetarian and Mixed Diet.

Mukkutrum:

In vali abanan,viyanan, devathathan and koorman, in azhal sathagam and alosagam and in iyyam santhigam were affected in most of the cases.

Udal Thathugal:

Saaram and oon were affected in all the patients.

Envagai Thervu:

Naadi and Moothiram were affected most of the patients.

Naadi :

Vatha pitha naadi was most common naadi felt.

Results after treatment:

55% of patients show good improvements, 30% of patients shows moderate improvement and in 15% of patients poor improvement was observed.

Pharmacological study reveals that Velvanga parpam contains Anti-tumour activity The antitumor effect of the drug is evident from the reduction in solid tumour volume and also the reversal of altered haematological parameter almost equal to normal. The drug can be used as a novel potential agent in the area of tumour chemotherapy.

The toxicity study revealed that there were no toxic effect was observed upto 5mg/kg of Velvanga Parpam on oral route over a period of 28 days.

The bio-statistical report of the clinical trial shows significant result.

Conclusion

CONCLUSION

- ✓ Ukkara soolai (Benign prostatic hyperplasia) is primarily due to the derangement of vatham.

- ✓ The trial medicine velvanga parpam predominating with Kaippu taste respectively Equivalent the vatham.

- ✓ From the pre clinical pharmacological studies it is evident that the medicines were significant Anti-tumour activity.

- ✓ The velvanga parpam did not produce any toxicity in lower dose in preclinical study. So it is safe drug for ukkara soolai (benign tumour).

- ✓ From the preclinical study of Velvanga parpam is reduction in solid tumour volume.

- ✓ In Bio-chemical analysis the trial medicine contains zinc and chloride. These are very essential for maintaining the prostate health.

- ✓ No contra indication was reported during the course of the treatment.

- ✓ The trial medicines gave maximum relief from the symptoms of Ukkara soolai.

- ✓ Therefore the authour concluded that the trial medicine velvanga parpam should be a very positive remedy for Ukkara soolai (BPH).

Annexures

Certificates

The Tamil Nadu Dr. M.G.R. Medical University

69, Anna Salai, Guindy, Chennai-600 032

This Certificate is awarded to Dr K. SAMRAJ

for participating as a ~~Resource Person~~ / Delegate in the VI Workshop on

"Research Methodology & Biostatistics"

for AYUSH Post-Graduates & Researchers

organized by the Department of Siddha

The Tamil Nadu Dr. M.G.R. Medical University

from 12th September 2011 to 16th September 2011

Dr. MAYILVAHANAN NATARAJAN
M.S.Orth. M.Ch.Orth. (L'pool) Ph.D. D.Sc. F.R.C.S. D.Sc. (Hon)^3
VICE CHANCELLOR

Dr. SUDHA SESHAYYAN, M.S.
REGISTRAR (FAC)

Dr. N. KABILAN, M.D. (Siddha)
READER, DEPT. OF SIDDHA

VEL'S COLLEGE OF PHARMACY

Approved by the Government of Tamil Nadu
Affiliated to The Tamil Nadu Dr. MGR Medical University
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E-mail : velscollege@gmail.com **Web site :** www.velscollege.com

S.No	Title of The Project	Name of The Investigator	Approval status/Remarks	Project Reference
22.	Antineoplastic activity –In-vivo cytotoxic activity of Velvanga parpam against various prostatic cancer	Dr. K.Samraj	Total number of animals proposed was 48 mice and after having discussion it was decided to reduce 8 number of animals.	XIII/VELS/PCOL/ 22/2000/CPCSEA/I AEC/11.08.2012
23.	Beneficial effects of Galangin c ethanol- induced inflammation pancreas- A study in rat model.	Fathima Cynthia Antony	Total number of animals proposed was 42 rats. All the 42 rats were Sanctioned.	XIII/VELS/PCOL/ 23/2000/CPCSEA/I AEC/08.08.12
24.	Role of Inflammasomes in rats subjected to experimental pancreatitis – Influence of β – sitosterol	P. Monika	Total number of animals proposed was 42 rats. All the 42 rats were Sanctioned.	XIII/VELS/PCOL/ 24/2000/CPCSEA/I AEC/08.08.12
25.	A study on antiulcer and wound healing property of a traditiona herbal preparation in rats.	Dr. J. Anbu	According to the protocol 46 rats were proposed, but only 36 rats were sanctioned.	XIII/VELS/PCOL/ 25/2000/CPCSEA/I AEC/08.08.12
26.	Anticonvulsant activity of Madhana Biravam and Raja Rajeswara Kuligai In animal models	Dr. A. Kirubagaran	Total number of animals sanctioned was 42 mice. it was advised to share the control and standard group results. Since the similar pattern of the study has been planned in the same department.	XIII/VELS/PCOL/ 26/2000/CPCSEA/I AEC/11.08.2012
27.	Spermatogenic activity of Isabg Chooranam in rats	Dr. Thillai Vaanan	Total number of animals proposed was 42 rats. But only 35 animals were sanctioned because, it was advised to share the control and standard group results.	XIII/VELS/PCOL/ 27/2000/CPCSEA/I AEC/11.08.2012

City Centre : No. 521/2, Anna Salai, (Opp. G.R. Complex), Nandanam, Chennai - 600 035.
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Department of Pharmacology & Toxicology
School of Pharmaceutical Sciences
Vels University
Pallavaram, Chennai-600 117.

Biochemical analysis

ANNEXURE-I

BIO-CHEMICAL ANALYSIS OF TRIAL MEDICINE

Preparation of Sodium Carbonate extract:

2 gm of the sample drug is mixed 5 gm of Sodium carbonate and taken in a 100 ml beaker and 20 ml of distilled water is added. The solution is boiled for 10 minutes, cooled and then filtered. The filtrate is called sodium carbonate extract.

S.No.	Experiment	Observation	Inference
1	**Test for Acid Radicals**		
a.	**Test for Sulphate** 2 ml of the above prepared extract is taken in a test tube. To this add 2ml of 4% Ammonium oxalate solution.	Absence of White Precipitate	Absent
b.	2ml of extract is added with 2ml of dilute hydrochloric acid until the effervescence ceases off. Then 2ml barium chloride solution is added.	Absence of White Precipitate	Absent
2.	**Test for Chloride:** 2ml of extract is added with dilute nitric acid till the effervescence ceases. Then 2ml of silver nitrate solution is added.	white precipitate obtained	Present

3.	**Test for Phosphate** 2ml of the extract is treated with 2 ml of Ammonium molybdate solution and 2ml of concentrated nitric acid.	Absence of Yellow Precipitate	Absent
4.	**Test for Carbonate:** 2ml of the extract is treated with 2ml of magnesium sulphate solution.	Absence of white precipitate	Absent
5.	**Test for Sulphide:** 1 gm of the substance is treated with 2ml of concentrated Hydrochloric acid	Absence of Rotten egg smelling	Absent
6.	**Test for Nitrate:** 1gm of the substance is heated with copper turnings and concentrated sulphuric acid and viewed the test tube vertically down.	Absence of reddish brown gas.	Absent
7. a.	**Test for Fluoride and oxalate** 2ml of the extract is added with 2ml of dilute acetic acid and 2ml of calcium chloride solution and heated.	Absence of white precipitate	Absent
b.	5 drops of clear solution is added with 2ml of dilute sulphuric acid and slightly warmed to this, 1 ml of dilute potassium permanganate solution is added.	Absence of KMNO4 solution discolourisation.	Absent
8.	**Test for Nitrite** 3 drops of the extract is placed on a filter paper. On that, 2 drops a Acetic Acid and 2 drops of Benzidine solution is placed.	Absence of yellowish red colour	Absent

9.	**Test for Borate** 2 pinches of the substance is made into paste by using Sulphuric acid and Alcohol (95%) and introduced into the blue flame.	Absence of Green tinged flame	Absent
II.	**TEST FOR BASIC RADICALS**		
10.	**Test for lead** 2 ml of the extract is added with 2 ml of Potassium iodide solution	Absence of Yellow precipitate	Absent
11a	**Test for Copper** One pinch of substance is made into paste with concentrated Hydrochloric acid in a watch glass and introduced into the non luminous part of the flame.	Absence of Bluish green coloured flame.	Absent
b.	2ml of the extract is added with excess of Ammonia solution	Absence of deep blue	Absent
12.	**Test for Aluminium** To the 2 ml of extract. Sodium Hydroxide solution is added in drops to excess.	Absence of White precipitate.	Absent
13a	**Test for Iron** To the 2 ml of extract, 2 ml of Ammonium Thiocyanate solution is added.	Absence of Blood red colour	Absent
b.	To the 2 ml of extract, 2 ml of Ammonium Thiocyanate solution and 2 ml of concentrated Nitric Acid is added.	Absence of Blood red colour.	Absent
14.	**Test for Zinc** To the 2 ml of extract Sodium Hydroxide solution is added in drops to excess.	White precipitate Obtained .	Present

15.	**Test for Calcium** 2 ml of the extract is added with 2 ml of 4% Ammonium Oxalate solution.	White precipitate Obtained	Present
16.	**Test for Magnesium** 2ml of extract, Sodium Hydroxide solution is added in drops to excess.	White precipitate Obtained	Present
17.	**Test for Ammonium** 2 ml of extract few ml of Nessler's Reagent and excess of Sodium Hydroxide solution are added.	Absence of Reddish brown precipitate .	Absent
18.	**Test for Potassium** A pinch of substance is treated with 2 ml of Sodium Nitrite solution and then treated with 2 ml of Cobal Nitrate in 30% glacial Acetic acid.	Absence of Yellow precipitate.	Absent
19.	**Test for Sodium** 2 pinches of the substance is made into paste by using Hydrochloric acid and introduced into the blue flame.	Absence of Yellow colour flame	Absent
20.	**Test for Mercury** 2 ml of the extract is treated with 2 ml of Sodium Hydroxide solution.	Absence of yellow precipitate	Absent
21.	**Test for Arsenic** 2 ml of extract is treated with 2 ml of silver Nitrate solution	Absence of Yellow precipitate.	Absent
22.	**Test for Starch** 2ml of extract is treated with weak iodine solution	Absence of Bluecolour.	Absent

23.	**Test of reducing Sugar** 5ml of Benedicts qualitative solution is taken in a test tube and allowed to boil for 2 minutes and added 10 drops of the extract and again boiled for 2 minutes. The colour changes are noted.	Absence of Green colour.	Absent
24.	**Test of the alkaliods** 2ml of the extract is treated with 2ml of potassium lodide solution.	Absence of Red colour	Absent
25.	**Test of the proteins** 2ml of the extract is treated with 2ml of 5% NaOH ,mix well and add 2 drops of copper sulphate solution.	Absence of Violet colour.	Absent

RESULTS:

The given sample contains.

Drug Name : Velvanga Parpam

a. Chloride

b. Zinc

c. Calcium

d. Magnesium.

Toxicological study

ANNEXURE-II

ACUTE AND SUB ACUTE TOXICITY STUDY ON VELVANGA PARPAM

Animals

Mice of either sex weighing 25-30g and rats weighing 210-240g were obtained from the animal house of Vels University. The animals were used with the approval of the Institute animal ethics committee and obtained from Vels University, Chennai. They were fed with a balanced standard pellet diet and maintained under standard laboratory conditions, providing 24-280C temperature, standard light cycle (12 h light, 12 h dark) and water ad libitum. Animals were kept in cages with raised floors of wide mesh to prevent coprophagy. Animal welfare guidelines were observed during the maintenance period and experimentation. The rats were randomly assigned to control and different treatment groups, six animals per group. The animals were acclimatized for one week under laboratory conditions.

ACUTE TOXICITY STUDY-OECD 425 GUIDELINES

Acute oral toxicity test for the Velvanga Parpam was carried out as per OECD Guidelines 425. As with other sequential test designs, care was taken to ensure that animals are available in the appropriate size and age range for the entire study. The test substance is administered in a single dose by gavage using a stomach tube or a suitable intubation cannula. The fasted body weight of each animal is determined and the dose is calculated according to the body weight. After the substance has been administered, food was withheld for a further 2 hours in mice.

The animals were observed continuously for the first 4 h and then each hour for the next 24 h and at 6 hourly intervals for the following 48 h after administering of the test drug, to observe any death or changes in general behaviour and other physiological activities. Single animals are dosed in sequence usually at 48 h intervals. However, the time interval between dosing is determined by the onset, duration, and severity of toxic signs. Treatment of an animal at the next dose was delayed until one is confident of survival of the previously dosed animal. General behavior, respiratory pattern, cardiovascular signs, motor activities, reflexes, change in skin and fur, mortality and the body weight changes were monitored daily. The time of onset, intensity, and duration of these signs, if any, was recorded.

SUB-ACUTE TOXICITY

In a 28-days sub acute toxicity study, twenty four either sex (3+3) rats were divided into four groups of 6 rats each. Group I that served as normal control was administered with distilled water (p.o.) while groups II, III and IV were administered daily with the Velvanga Parpam (p.o.) for 28 days at a dose of 2.5, 5.0 and 10mg/kg respectively. The animals were then observed daily for gross behavioural changes and any other signs of subacute toxicity. The weight of each rat was recorded on day 0 and weekly throughout the course of the study, food and water consumption per rat was calculated. At the end of the 28 days they were fasted overnight, each animal was anaesthetized with diethylether, following which they were then dissected and blood samples were obtained by cardiac puncture into heparinised tubes. The blood sample

collected from each rat was centrifuged with 3000 X g at 4oC for 10 min to separate the serum and used for the biochemical assays.

Hematological and blood biochemical analyses:

At the end of the study, all animals were kept fasted for 16-18 h and then anesthetized with anesthetic ether on the 28th day. Blood samples for hematological and blood chemical analyses were taken from retro orbital vein. Heparinized blood samples were taken for determining complete blood count (white blood cell count, differential white blood cell count, platelet count, red blood cell count, hematocrit, and hemoglobin) by semiautomated hematology analyzer. The serum from non-heparinized blood was carefully collected for blood chemistry and enzyme analysis (glucose, creatinine, total protein, albumin, total and direct bilirubins, serum glutamate-oxaloacetate transaminase (SGOT), serum glutamate pyruvate transaminase (SGPT), and alkaline phosphatase (ALP)) were automatically determined using autoanalyzer.

Necropsy:

All rats were sacrificed after the blood collection. The positions, shapes, sizes and colors of internal organs were evaluated. The Spleen, Testes, Pancrea, Lung, Liver, Brain, Heart, Stomach, Intestine, Bone, Ovary, and Kidney tissues were excised from all rats to visually detect gross lesions, and weighed to determine relative organs' weights and preserved in 10% neutral formalin for histopathological assessment. The tissues were embedded in paraffin, and then sectioned, stained with haematoxylin and eosin and were examined microscopically.

Statistical analysis

Values were represented as mean ± SEM. Data were analysed using one-way analysis of variance (ANOVA) using GraphPad Instat-V3 software. P values < 0.05 were considered significant.

RESULTS

All the animals from control and all the treated dose groups up to 5mg/kg survived throughout the dosing period of 28 days. No signs of major or significant intoxication were observed in animals from lower to higher dose groups during the dosing period of 28 days. Animals from all the treated dose groups exhibited comparable body weight gain with that of controls throughout the dosing period of 28 days.

Food consumption of control and treated animals was found to be comparable throughout the dosing period of 28days. Ophthalmoscopic examination, conducted prior to and at the end of dosing period on animals from control and all the treated dose groups did not reveal any abnormality. The results of haematological investigations revealed following significant changes in the values of different parameters investigated when compared with those of respective controls; However, the increase or decrease in the values obtained was within normal biological and laboratory limits.

A slight decrease in total RBC count values were obtained for animals in the dose group of 2.5 and 5mg/kg ($P<0.05$). Decreased values of platelets ($P<0.05$) were observed for animals in dose groups administered 5-10mg/ kg body weight of Velvanga Parpam

sacrificed on day 28. Results of Biochemical investigations conducted on days 28 and revealed the following significant changes in the values of different parameters studied when compared with those of respective controls; however, the values obtained were within normal biological and laboratory limits.

Protein level is elevated in animals of 2.5 and 5mg/kg dose group ($P<0.05$). Aspartate Amino transferase levels slightly decreased in animals of 5 and 10mg/kg group ($P<0.01$). 8) Functional observation tests conducted at termination revealed no abnormalities. Urine analysis, conducted at the end of the dosing period in week 4 revealed no abnormality attributable to the treatment. Organ weight data of animals sacrificed at the end of the dosing period was found to be comparable with that of respective controls. Gross pathological examination did not reveal any abnormality. Histopathological examination did not reveal any abnormality.

CONCLUSION:

Based on these findings, no toxic effect was observed upto 5mg/kg of Velvanga Parpam on oral route over a period of 28 days. So, it can be concluded that the Velvanga Parpam can be prescribed for therapeutic use in human with the dosage recommendations of upto 5mg/kg. body weight p.o.

REFERENCES

1. Benjamin, M.N., 1978.Outline of Veterinary Clinical Pathology. University Press, IOWA, USA., pp:229-232.

2. OECD (testing guideline, 407), 1995. Repeat dose 28 days oral toxicity study in rodents; In Guidance document for the development of OECD guideline for testing of chemicals Environmental monographs No 76; http//www.oecd.ord/document/30/0.2340,en??2649-34377-19166381111, 00html.

3. Organization for Economic Cooperation Development (OECD) Guideline, 425,2000. Guideline Document on Acute Oral Toxicity. Environmental Health and Safety Monograph Series on Testing and Assessment No. 24.

4. Ringler, D.H. and L.Dabich, 1979. Haematology and Clinical Biochemistry. In: The Laboratory Rat. Baker, J., J.R. Lindsey and S.H.Weisbroth (Eds.), Academic Press London, 1: 105-118.

Table 1: Dose finding experiment and its behavioral Signs of Toxicity

No	Dose mg/kg	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20
1.	**50**	+	-	-	+	-	+	+	-	-	-	-	-	-	-	-	-	-	-	+	-
2	**300**	+	+	-	+	-	+	+	+	+	-	-	-	-	-	-	+	-	+	+	+
3	**2000**	+	+	-	+	-	+	+	+	+	+	-	-	+	-	-	+	-	+	+	+

1. Alertness 2. Aggressiveness 3. Pile erection 4. Grooming 5. Gripping 6. Touch Response 7. Decreased Motor Activity 8. Tremors 9. Convulsions 10. Muscle Spasm 11. Catatonia 12. Muscle relaxant 13. Hypnosis 14. Analgesia 15.Lacrimation 16. Exophthalmos 17. Diarrhoea 18. Writhing 19. Respiration 20. Mortality

Table 2. Body wt (g) of albino rats exposed to *Velvanga Parpam* for 28days.

Dose (mg/kg/day)	Days				
	1	**7**	**14**	**21**	**28**
Control	210.11±5.00	212.15±4.05	214.25±5.20	218.22±6.12	221.00±5.00*
2.5	215.00±4.31	217.12±4.52	220.31±4.16*	222.64±8.21**	224.62±6.10*
5.0	214.13±5.00	214.10±4.82	211.00±3.18	210.10±5.00	207.72±4.21*
10	212.32±5.20	210.20±5.40	210.12±5.24	205.02±4.38	204.12±3.12*

Values are mean of 6 animals ± S.E.M. *P<0.05; **P<0.01.

Table 3. Food (g/day) intake of albino rats exposed to *Velvanga Parpam* for 28days.

Dose (mg/kg/day)	Days (gms/rats)				
	1	7	14	21	28
Control	45.00±2.75	44.54±2.18	46.45±2.19	45.00±2.58	47.50±3.42
2.5	44.21±2.48	45.43±2.42	46.24±2.46	49.12±2.49	48.18±3.00
5.0	42.35±2.10	42.00±2.54	44.20±2.42	45.81±3.56	46.40±3.00
10	43.43±2.61	45.24±2.80	44.11±2.80	45.19±2.02	45.06±3.11

Values are mean of 6 animals ± S.E.M. $^{ns}P>0.05$Vs control.

Table 4. Water (ml/day) intake of male and female albino rats exposed to *Velvanga Parpam* for 28days.

Dose (mg/kg/day)	Days(ml/rat)				
	1	7	14	21	28
Control	52.04±2.80	52.02±3.43	55.21±3.15	50.13±3.14	50.24±3.20
2.5	52.28±2.42	51.20±3.04	48.20±4.02	46.18±3.00	42.52±2.48*
5.0	48.14±2.18	50.14±3.72	44.28±3.34	42.12±2.92	44.14±3.37
10	50.43±3.52	52.72±3.00	50.22±3.80	46.42±3.12	44.32±3.15

Values are mean of 6 animals ± S.E.M. *P<0.05; Vs control.

Table 5. Hematological parameters after 28days treatment with *Velvanga Parpam* in rats.

Parameter	Control	2.5 mg/kg	5 mg/kg	10 mg/kg
Red blood cell (mm^3)	8.18±0.72	7.59±0.44	7.25±0.52	7.13±0.50
HB (%)	14.42±0.30	12.10±0.35	12.51±0.40	11.02±0.44*
Leukocyte ($x10^6$/mL)	10215±112.55	10415±215.14	10206±224.11	10346±212.00
Platelets/ul	1440±34.10	1392±32.12	1172±30.22**	998±23.16**
MCV (gl)	55.62±5.42	55.10±5.72	55.45±5.22	56.18±4.62
Neutrophil	5.54±1.43	5.22±1.20	4.84±0.92*	5.14±3.27
Lymphocyte	92.32±2.90	91.48±3.12	93.20±3.24	94.30±3.86
Monocyte	2.15±0.30	2.31±0.34	2.25±0.22	2.28±0.31
Eosinophil	1.00±0.00	1.0±0.22	1.0±0.11	1.00±0.12
Basophil	0	0	0	0
ESR(mm)	1±00	1±00	1±00	1±00
PCV	42.30±2.52	45.11±2.18	45.52±3.04	45.42±3.00

Values are mean of 6 animals ± S.E.M. *P<0.05; **P<0.01. Vs control.

Table 6. Effect of treatment with *Velvanga Parpam* biochemical parameters.

Dose (mg/kg)	Control	2.5 mg/kg	5 mg/kg	10 mg/kg
Total Bilirubin (mg/dL)	0.200±0.05	0.220±0.06**	0.225±0.05**	0.218±0.04**
Bilirubin direct (mg/dL)	0.1±0.04	0.1±0.05	0.1±0.04	0.1±0.05
Bilirubin indirect(mg/dL)	0.1±00	0.1±00	0.1±00	0.1±00
ALP (U/L)	380.32±10.10	414.20±12.13**	456.82±10.02	494.21±12.22
SGOT (U/L)	176.21±5.18	160.26±6.52**	156.23±5.10*	154.12±5.50*
SGPT(U/L)	45.2±2.32	44.18±3.22	45.83±2.52	44.60±4.17
Total Protein(g/dl)	9.02±1.22	8.07±0.30*	8.15±0.27*	8.10±0.42*
Albumin(g/dl)	3.17±0.25	3.10±0.24	3.16±0.23	3.10±0.22
Globulin(g/dl)	5.00±0.18	4.18±0.22*	4.28±0.24*	4.28±0.23*

Values are mean of 6 animals ± S.E.M. *P<0.05; **P<0.01. Vs control.

Table-7 RFT

Dose (mg/kg)	Control	2.5 mg/kg	5 mg/kg	10 mg/kg
Urea(mg/dL)	55.40±2.35	54.32±3.62	55.42±2.18	55.82±2.30
Creatinine (mg/dL)	0.77±0.05	0.76±0.05	0.78±0.06	0.76±0.05
Uric acid (mg/dL)	1.62±0.12	1.16±0.18**	1.26±0.16*	1.06±0.12**
Na m.mol	142.80±5.22	144.5±5.00	142.12±5.22	140.28±5.10
K m.mol	20.45±2.48	19.40±2.60	20.05±2.42	20.18±2.02
Cl m.mol	100.25±4.46	100.20±5.22	99.78±4.72	100.02±4.10

Values are mean of 6 animals ± S.E.M. *P<0.05; **P<0.01. Vs control.

Table-8. Lipid Profile

Dose (mg/kg)	Control	2.5 mg/kg	5 mg/kg	10 mg/kg
Total cholestrol(mg/dL)	40.82±2.52	41.10±2.42	40.28±3.24	41.00±3.01
HDL(mg/dL)	13.02±1.42	13.20±1.47	13.20±2.42	13.24±2.23
LDL(mg/dL)	44.00±2.80	44.05±3.60	43.38±3.20	44.22±3.20
VLDL(mg/dl)	16.32±2.60	15.22±2.42	16.10±1.42	15.00±1.14
Triglycerides (mg/dl)	86.04±3.02	85.18±2.22	86.32±3.40	85.14±2.72
TC/HDL ratio (g/dl)	3.66±0.25	3.70±0.28	3.70±0.30	3.52±0.28
Blood glucose(mg/dl)	125.30±6.47	126.05±5.20	126.15±5.62	125.21±2.57

Values are mean of 6 animals ± S.E.M. $^{ns}P>0.05$; Vs control.

Table-9 Urine Analysis

Parameters	Control	2.5 mg/kg	5 mg/kg	10 mg/kg
Colour	Yellow	Yellow	Yellow	Yellow
Transparency	Clear	Slightly turbid	Slightly cloudy	Slightly turbid
Specific gravity	1.010	1.010	1.010	1.010
PH	>7.2	>8.0	>8.0	>9.0
Protein	Nil	3+	3+	3+
Glucose	Nil	Nil	Nil	Nil
Bilirubin	-ve	-ve	-ve	-ve
Ketones	-ve	+ve	+ve	+ve
Blood	Absent	Absent	Absent	Absent
Urobilinogen	Normal	Abnormal	Abnormal	Abnormal
Pus cells	0-cells/HPF	1-cell/HPF	2-cells/HPF	1-cell/HPF
RBCs	Nil	Nil	0-1cells/HPF	Nil
Epithelial cells	Nil	1-cell/HPF	Nil	1-cell/HPF
Crystals	Nil	Nil	Nil	Nil
Casts	Nil	Nil	Nil	Nil
Others	Bacteria seen	Bacteria seen	Bacteria seen	Bacteria seen

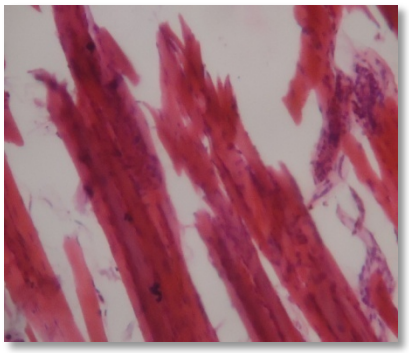
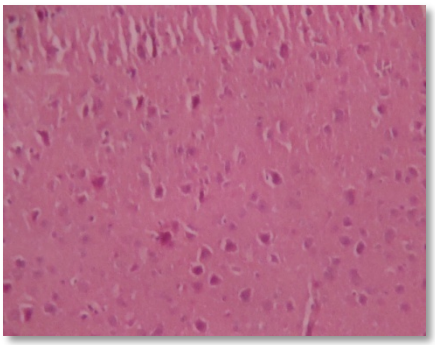
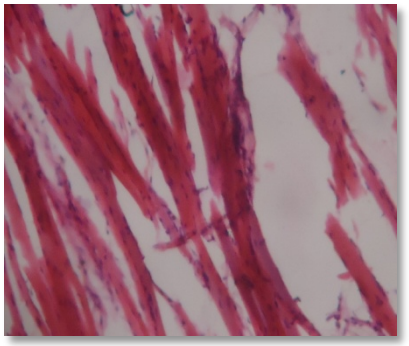
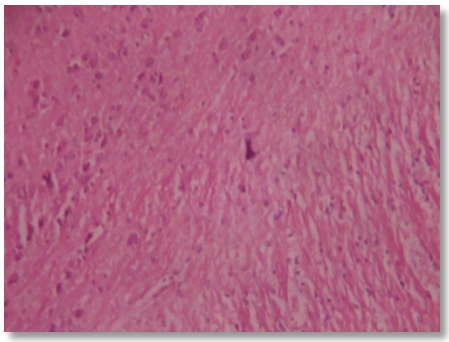
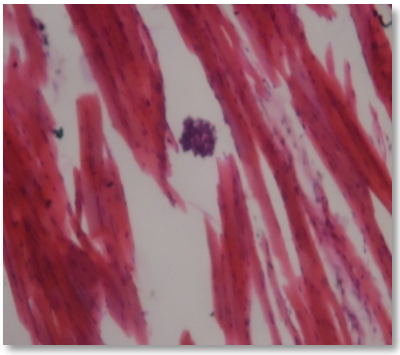
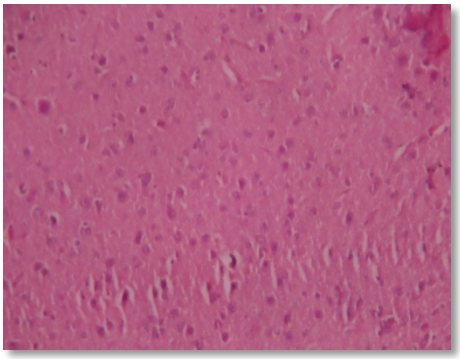
Table 10. Effect of oral administration of a *Velvanga Parpam* on organ weight

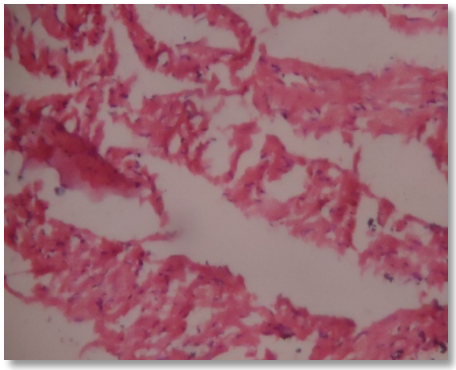
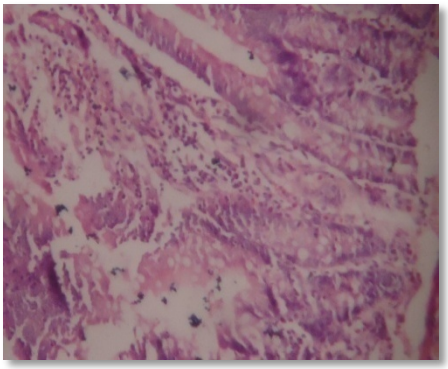
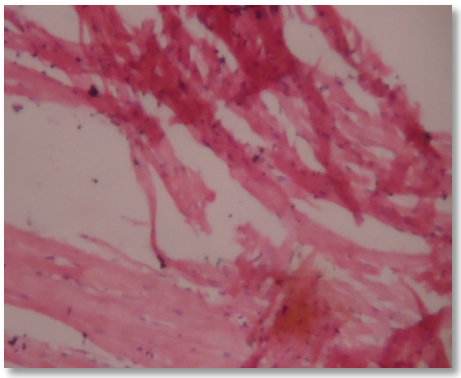
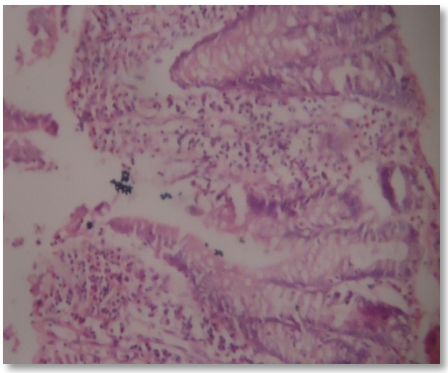
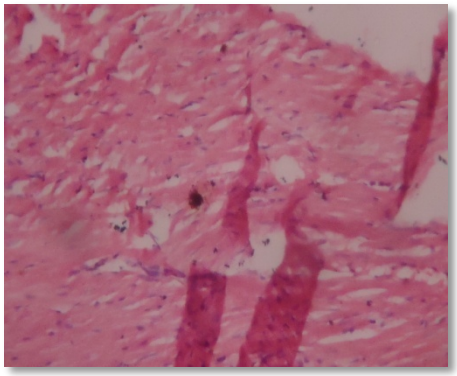
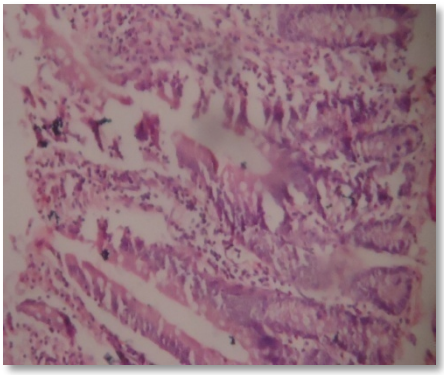
Dose (mg/kg)	Control	2.5 mg/kg	5 mg/kg	10 mg/kg
Liver (g)	5.27±0.17	5.00±0.15	4.82±0.12	4.71±0.18*
Heart (g)	0.62±0.04	0.60±0.05	0.58±0.04	0.58±0.04
Lung (g)	1.45±0.06	1.44±0.14	1.46±0.24	1.50±0.15**
Spleen (g)	0.65±0.05	0.65±0.04	0.66±0.04	0.65±0.05
Ovary (g)	1.71±0.14	1.73±0.15	1.70±0.18	1.72±0.15
Testes (g)	1.48±0.10	1.45±0.12	1.46±0.15	1.46±0.15
Brain (g)	1.56±0.15	1.58±0.13	1.56±0.14	1.53±0.14
Kidney (g)	0.73±0.04	0.71±0.04	0.70±0.04	0.72±0.05
Stomach (g)	1.36±0.14	1.35±0.12	1.36±0.11	1.35±0.15

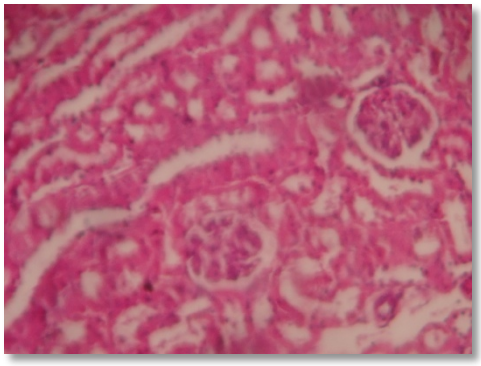
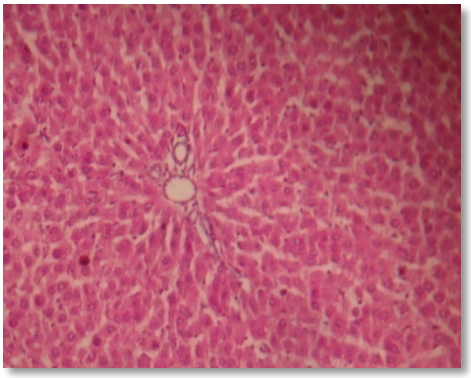
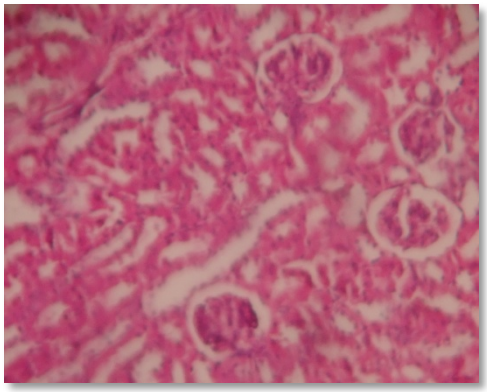
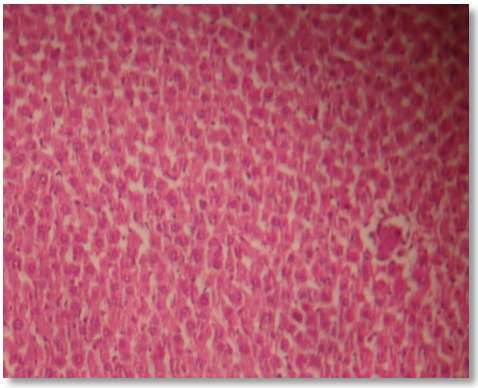
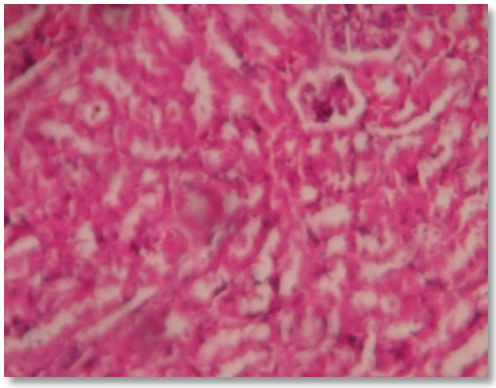
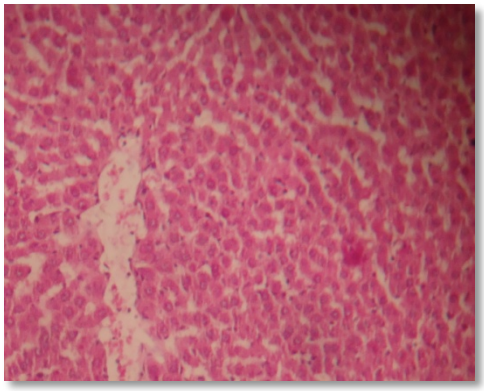
Values are mean of 6 animals ± S.E.M. *P<0.05; **P<0.01 Vs control.

DRUG OF VELVANGA PARPAM

HISTOPATHOLOGY

	BONE	BRAIN
2.5 mg		
5 mg		
10 mg		

	HEART	INTESTINE
2.5 mg		
5 mg		
10 mg		

	KIDNEY	LIVER
2.5 mg		
5 mg		
10 mg		

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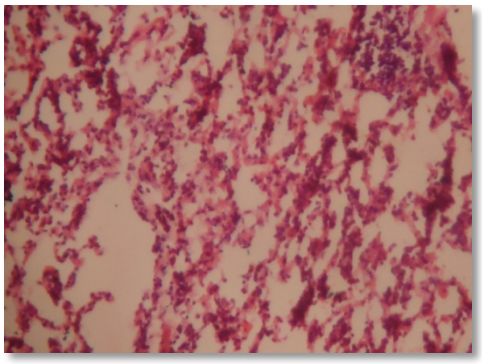
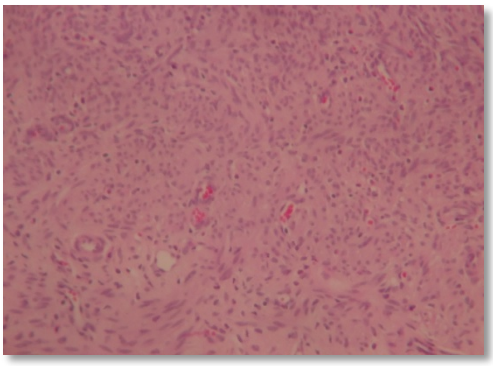
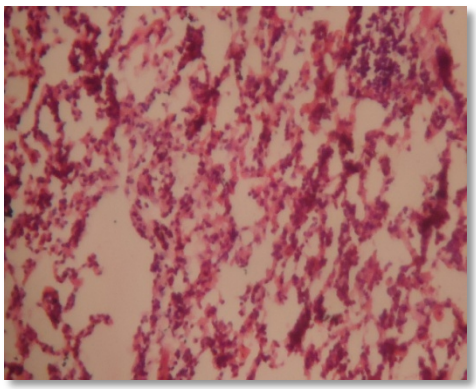
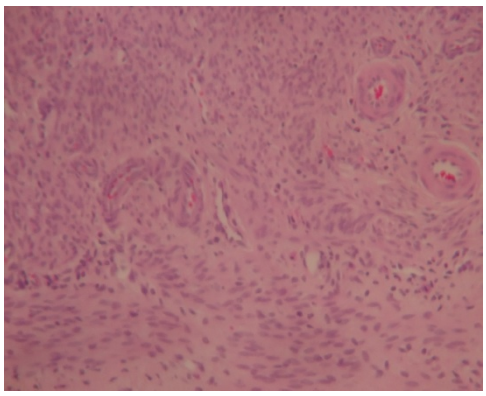
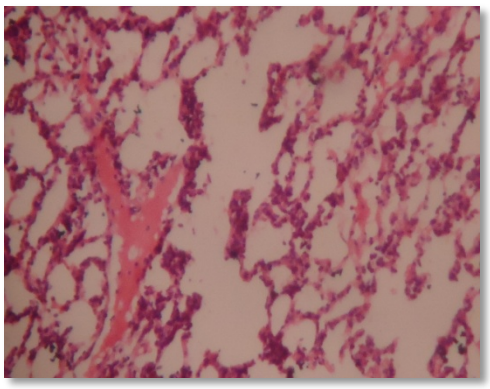
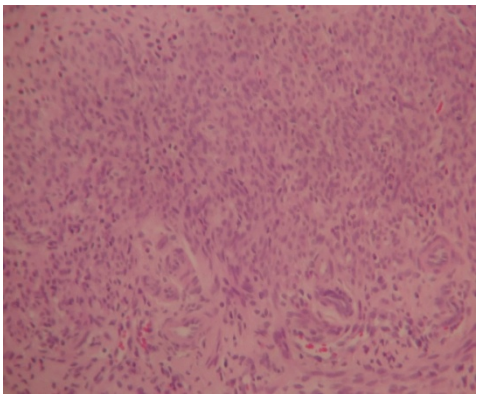
2.5 mg		
5 mg		
10 mg		

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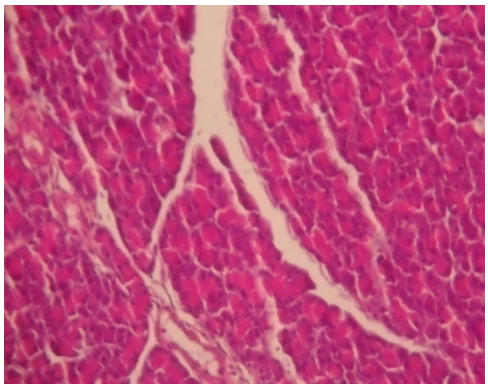
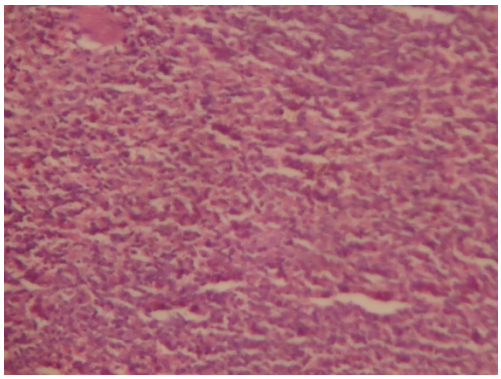
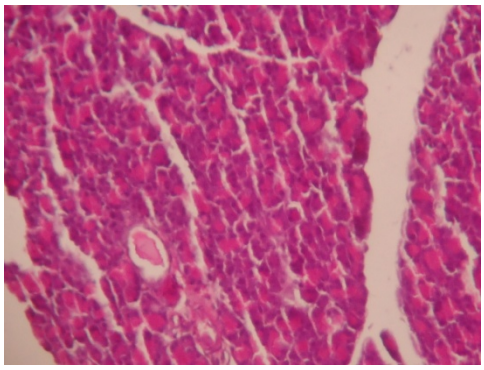
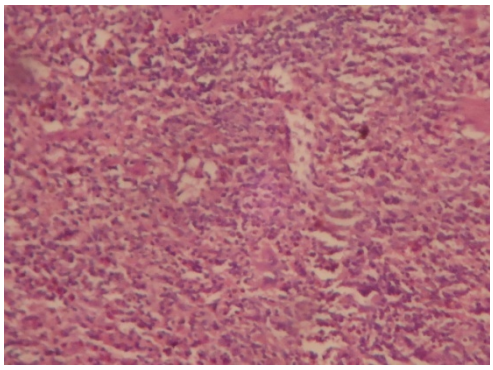
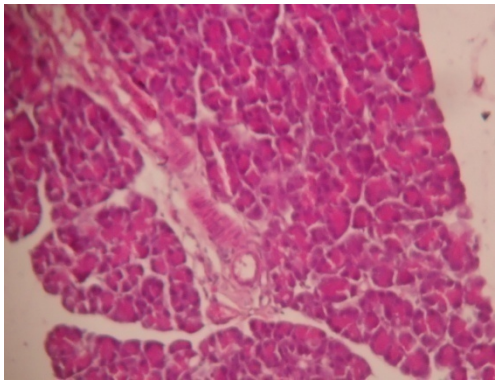
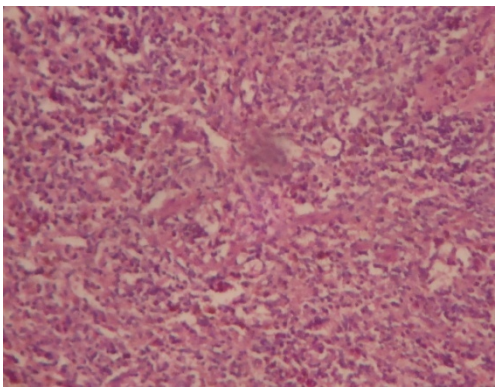
2.5 mg		
5 mg		
10 mg		

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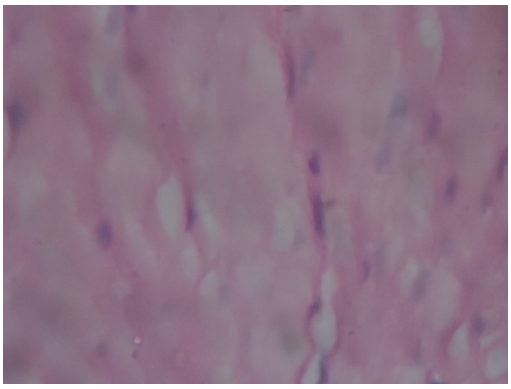
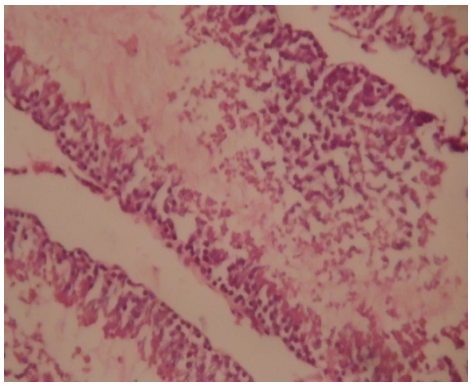
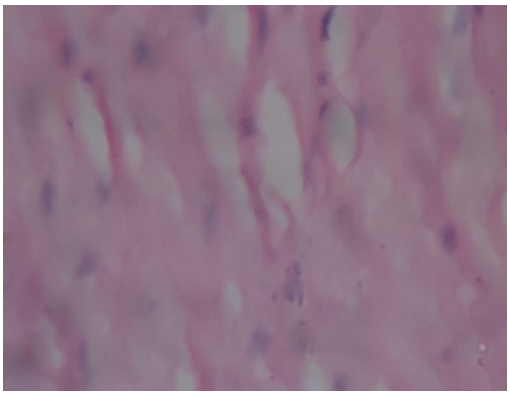
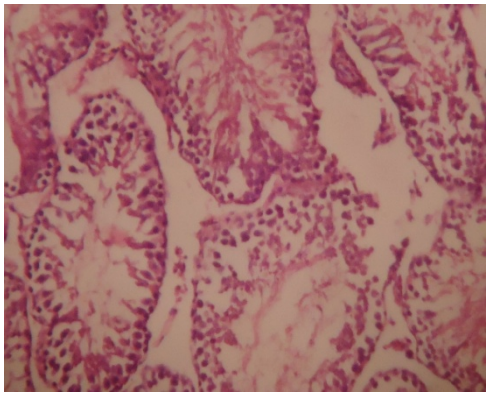
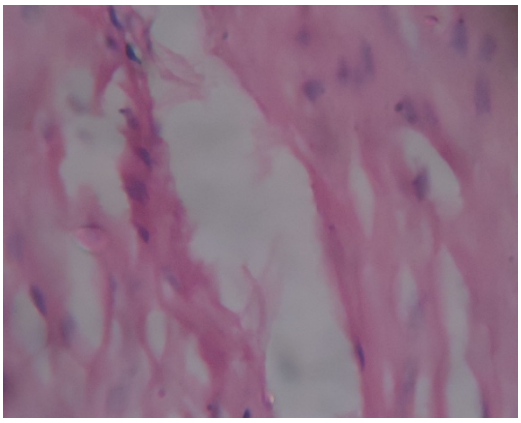
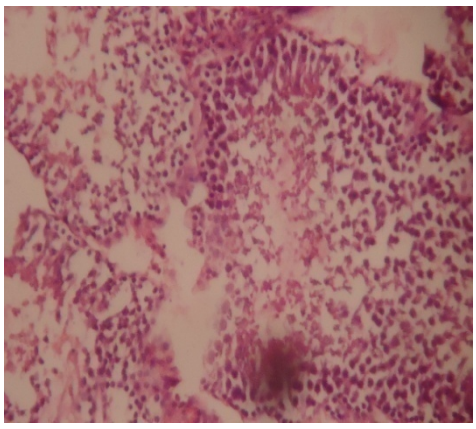
2.5 mg		
5 mg		
10 mg		

Pharmacological study

ANNEXURE- III

ANTITUMOR ACTIVITY OF VELVANGA PARPAM IN RODENTS

INTRODUCTION:

In most people's minds there is no scarier diagnosis than that of cancer. Cancer is often thought of as an untreatable, unbearably painful disease with no cure. However popular this view of cancer may be, it is exaggerated and over-generalized. Cancer is undoubtedly a serious and potentially life-threatening illness. The truth of the matter is that there are multiple types of cancer, many of which can today be effectively treated so as to eliminate, reduce or slow the impact of the disease on patients' lives. While a diagnosis of cancer may still leave patients feeling helpless and out of control, in many cases today there is cause for hope rather than hopelessness. Normal cells in the body grow and divide for a period of time and then stop growing and dividing. Thereafter, they only reproduce themselves as necessary to replace defective or dying cells. Cancer occurs when this cellular reproduction process goes out of control. In other words, cancer is a disease characterized by uncontrolled, uncoordinated and undesirable cell division. Unlike normal cells, cancer cells continue to grow and divide for their whole lives, replicating into more and more harmful cells. The abnormal growth and division observed in cancer cells is caused by damage in these cells' DNA (genetic material inside cells that determines cellular characteristics and functioning). There are a variety of ways that cellular DNA can become damaged and defective. Alternatively, defective DNA can be inherited from your parents. As cancer cells divide and replicate themselves, they often form into a clump of cancer cells known as a tumor. Tumors cause many of the symptoms of cancer by pressuring, crushing and destroying surrounding non-cancerous cells and tissues. Tumors come in two forms; benign and malignant. Benign tumors are

not cancerous, thus they do not grow and spread to the extent of cancerous tumors. Benign tumors are usually not life threatening. Malignant tumors, on the other hand, grow and spread to other areas of the body. The process whereby cancer cells travel from the initial tumor site to other parts of the body is known as metastasis. The causes of cancer are not fully understood, but years of research have brought to light risk factors that increase people's chances of getting particular types of cancer. Some of these risk factors are inevitable, while others can be avoided by choosing to live a healthy lifestyle.

The Normal Prostate Gland

The prostate is a walnut-sized gland that forms part of the male reproductive system. The gland is made of two lobes, or regions, enclosed by an outer layer of tissue. As the diagrams show, the prostate is located in front of the rectum and just below the bladder, where urine is stored. The prostate also surrounds the urethra, the canal through which urine passes out of the body. Scientists do not know all the prostate's functions. One of its main roles, though, is to squeeze fluid into the urethra as sperm move through during sexual climax. This fluid, which helps make up semen, energizes the sperm and makes the vaginal canal less acidic.

The normal prostate is composed of glands and stroma. The glands are seen in cross section to be rounded to irregularly branching. These glands represent the terminal tubular portions of long tubuloalveolar glands that radiate from the urethra. The glands are lined by two cell layers: an outer low cuboidal layer and an inner layer of tall columnar mucin-secreting epithelium. These cells project inward as papillary projections. The fibromuscular stroma between the glands accounts for about half of the volume of the prostate.

Prostatitis

Acute prostatitis is not common, but is most likely to occur in young men. Causative agents include bacterial organisms similar to those causing urinary tract infections, as well as Neisseria gonorrheae. A related complication of prostatic abscess is

uncommon. The and slight enlargement of the prostate with acute inflammation may cause acute rectal, lower back, or perineal pain along with fever. There can be dysuria. The prostate is enlarged and tender. Urine culture may be done, but prostatic massage is contraindicated. Microscopically, the glands are filled with neutrophils, and the intervening stroma may also contain a few neutrophils, explaining the presence of neutrophils on urine microscopic examination.

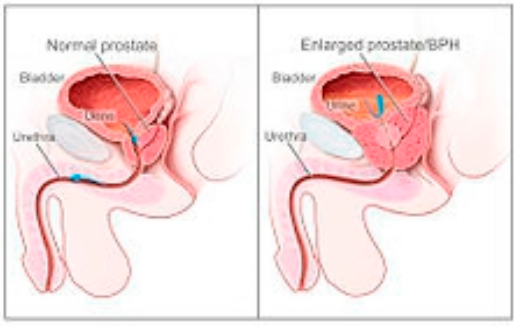
Chronic prostatitis may follow acute prostatitis, but is more likely to occur without prior history in older men, and may suggest an underlying obstructive urinary tract abnormality. There can be intermittent urinary frequency and dysuria. The prostate may not be enlarged. Prostatic massage may increase the yield of urine culture. Routine cultures, however, do not identify one common organism: Ureaplasma urealyticum.

Chronic abacterial prostatitis is the most common cause for prostatitis, but is difficult to diagnose from lack of specific findings. No organism can be identified as a causative agent. Symptoms of dysuria along with low grade pelvic pain or low back pain may be present. Microscopically, lymphocytes, plasma cells, and macrophages appear in the prostatic stroma. Prostatitis can elevate the serum prostate specific antigen, but generally not more than double normal, and generally not increasing significantly over time.

Prostatic Hyperplasia

Nodular prostatic hyperplasia (benign prostatic hyperplasia, or BPH) is a common condition as men age. The mechanism for hyperplasia may be related to accumulation of dihydrotestosterone in the prostate, which then binds to nuclear hormone receptors which then trigger growth. The normal prostate weighs 20 to 30 gm, but most prostates with nodular hyperplasia can weigh from 50 to 100 gm.

Diagram illustrating normal prostate (left) and benign prostatic hyperplasia (right).



Prevalence and Incidence of BPH

Benign prostatic hyperplasia (BPH) is the proliferation of nonmalignant stromal and epithelial cells in the prostate, which may lead to nodular formation in the periurethral area of the prostate and subsequent partial or complete obstruction of the urethra. Clinical BPH is a diagnosis of lower urinary tract symptoms (LUTS), urinary tract infections (UTIs), or acute urinary retention due to the urethral obstruction. BPH progresses linearly with age. According to the National Institutes of Health, there are more than 7.8 million BPH diagnoses made. Histological evidence of BPH emerges after age 30, with 50% prevalence in men age 50-61 and 90% prevalence by age 90. However, it is difficult to predict how many of these cases will progress to clinical BPH. The overall prevalence of clinical BPH (BPH with LUTS) is 10.3%, with a maximum prevalence of 24% by age 80. It is estimated that 45% of nonsymptomatic 46-year-old men with histological BPH will develop LUTS over the next 30 years.

The cause of BPH is not well understood. No definite information on risk factors exists. Theory focuses on dihydrotestosterone (DHT), a substance derived from testosterone prostate, which may help control its growth. Most animals lose their ability to produce DHT as they age. However, some research has indicated that even with a drop

in the blood's testosterone level, older men continue to produce and accumulate high levels of DHT in the prostate. This accumulation of DHT may encourage the growth of cells. Scientists have also noted that men who do not produce DHT do not develop BPH.

Signs and Symptoms of BPH

Symptoms

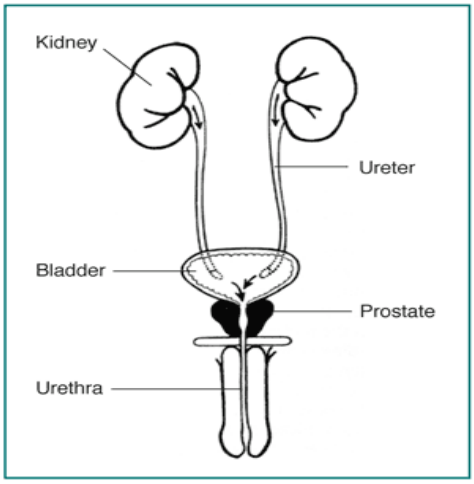
Screening and diagnostic procedures for BPH are similar to those used for prostate cancer. Some signs to look for include:

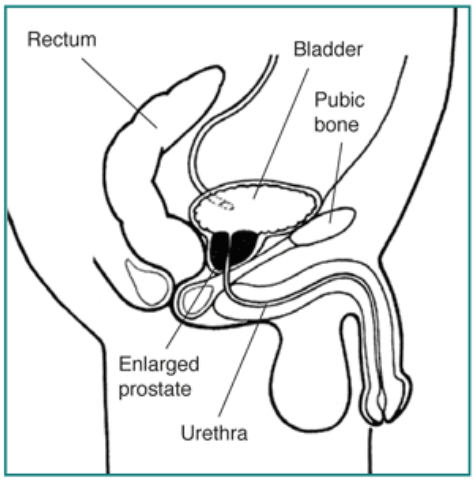
- Weak urinary stream
- Prolonged emptying of the bladder
- Abdominal straining
- Hesitancy
- Irregular need to urinate
- Incomplete bladder emptying
- Post-urination dribble
- Irritation during urination
- Frequent urination
- Nocturia (need to urinate during the night)
- Urgency
- Incontinence (involuntary leakage of urine)
- Bladder pain
- Dysuria (painful urination)

- Problems in ejaculation

The size of the prostate does not always determine how severe the obstruction or the symptoms will be. Some men with greatly enlarged glands have little obstruction and few symptoms while others, whose glands are less enlarged, have more blockage and greater problems. In most males, the prostate gland slowly begins to enlarge around the age of 30, but it is usually asymptomatic until age 50. An enlarged prostate can cause both obstructive (voiding) and irritative (holding) symptoms that decrease a patient's quality of life. BPH may lead to UTIs and AUR, which may require surgical intervention, but these are often preceded by LUTS. Obstructive symptoms include decreased force of the urinary stream, straining to urinate and hesitancy to initiate urination, interruptions in flow, and the feeling of not emptying the bladder. Irritative symptoms include frequency, nocturia, dysuria, urgency, and urge incontinence. These symptoms may decrease a patient's quality of life by interfering with his ability to perform his job, to travel, to attend social engagements, and to get a good night's sleep. Depending on the individual, only some of these symptoms may be present, and to varying degrees of severity.

A digital rectal exam may reveal an enlarged prostate. An elevated PSA level may also signify an enlarged prostate, among other possible diagnoses, and should be followed by a free PSA determination.





Normal urine flow **Urine flow with BPH.**

As a man matures, the prostate goes through two main periods of growth. The first occurs early in puberty, when the prostate doubles in size. At around age 25, the gland begins to grow again. This second growth phase often results, years later, in BPH. Though the prostate continues to grow during most of a man's life, the enlargement doesn't usually cause problems until late in life. BPH rarely causes symptoms before age 40, but more than half of men in their sixties and as many as 90 percent in their seventies and eighties have some symptoms of BPH.

As the prostate enlarges, the layer of tissue surrounding it stops it from expanding, causing the gland to press against the urethra like a clamp on a garden hose. The bladder wall becomes thicker and irritable. The bladder begins to contract even when it contains small amounts of urine, causing more frequent urination. Eventually, the bladder weakens and loses the ability to empty itself, so some of the urine remains in the bladder. The narrowing of the urethra and partial emptying of the bladder cause many of the problems associated with BPH.

Sometimes a man may not know he has any obstruction until he suddenly finds himself unable to urinate at all. This condition, called acute urinary retention, may be triggered by taking over-the-counter cold or allergy medicines.

Such medicines contain a decongestant drug, known as a sympathomimetic. A potential side effect of this drug may prevent the bladder opening from relaxing and allowing urine

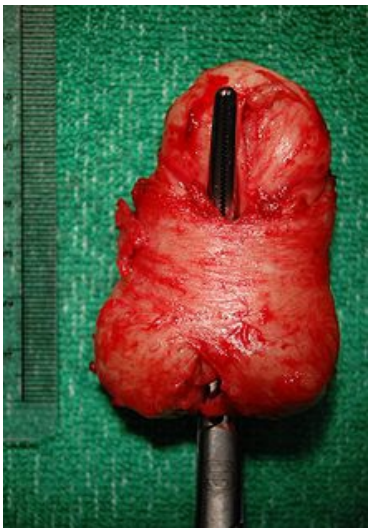
to empty. When partial obstruction is present, urinary retention also can be brought on by alcohol, cold temperatures, or a long period of immobility.

It is important to tell your doctor about urinary problems such as those described above. In eight out of 10 cases, these symptoms suggest BPH, but they also can signal other, more serious conditions that require prompt treatment. These conditions, including prostate cancer, can be ruled out only by a doctor's examination.

Severe BPH can cause serious problems over time. Urine retention and strain on the bladder can lead to urinary tract infections, bladder or kidney damage, bladder stones, and incontinence-the inability to control urination. If the bladder is permanently damaged, treatment for BPH may be ineffective. When BPH is found in its earlier stages, there is a lower risk of developing such complications.

Diagnosis

Prostate with a large median lobe bulging upwards.



When BPH is suspected, it may be referred to urologist, a doctor who specializes in problems of the urinary tract and the male reproductive system. Several tests help the doctor identify the problem and decide whether surgery is needed. The tests vary from patient to patient, but the following are the most common.

Digital Rectal Examination

This examination is usually the first test done. The doctor inserts a gloved finger into the rectum and feels the part of the prostate next to the rectum. This examination gives the doctor a general idea of the size and condition of the gland.

Prostate-Specific Antigen (PSA) Blood Test

To rule out cancer as a cause of urinary symptoms, doctor may recommend a PSA blood test. PSA, a protein produced by prostate cells, is frequently present at elevated levels in the blood of men who have prostate cancer. Prostate cancers may be detected by digital examination, by ultrasonography (transrectal ultrasound), or by screening with a blood test for prostate specific antigen (PSA). None of these methods can reliably detect all prostate cancers, particularly the small cancers. Widespread PSA screening is not cost-effective. PSA is a glycoprotein produced almost exclusively in the epithelium of the prostate gland. In the circulation PSA may be complexed to serum proteins (complexed PSA, or cPSA) or may be free (fPSA). The cPSA and fPSA together comprise total PSA (tPSA).

The tPSA is normally less than 4 ng/mL. A mildly increased tPSA in a patient with a very large prostate can be due to nodular hyperplasia, or to prostatitis. The fPSA correlates more closely with benign prostatic conditions than the tPSA. Men who have findings suspicious for carcinoma on digital rectal examination and a tPSA of <4 ng/mL have a probability of cancer of at least 10%, while those with tPSA levels from 4 to 10 ng/mL have a 25% probability. Men with tPSA's above 10 ng/mL have a >50% likelihood of having a prostate cancer.

Rectal Ultrasound and Prostate Biopsy

If there is a suspicion of prostate cancer, your doctor may recommend a test with rectal ultrasound. In this procedure, a probe inserted in the rectum directs sound waves at the prostate. The echo patterns of the sound waves form an image of the prostate gland on

a display screen. To determine whether an abnormal-looking area is indeed a tumor, the doctor can use the probe and the ultrasound images to guide a biopsy needle to the suspected tumor. The needle collects a few pieces of prostate tissue for examination with a microscope.

Urine Flow Study

Doctor may ask patient to urinate into a special device that measures how quickly the urine is flowing. A reduced flow often suggests BPH.

Cystoscopy

In this examination, the doctor inserts a small tube through the opening of the urethra in the penis. This procedure is done after a solution numbs the inside of the penis so all sensation is lost. The tube, called a cystoscope, contains a lens and a light system that help the doctor see the inside of the urethra and the bladder. This test allows the doctor to determine the size of the gland and identify the location and degree of the obstruction.

Treatment

Men who have BPH with symptoms usually need some kind of treatment at some time. However, a number of researchers have questioned the need for early treatment when the gland is just mildly enlarged. The results of their studies indicate that early treatment may not be needed because the symptoms of BPH clear up without treatment in as many as one-third of all mild cases.

Instead of immediate treatment, they suggest regular checkups to watch for early problems. If the condition begins to pose a danger to the patient's health or causes a major inconvenience to him, treatment is usually recommended.

Since BPH can cause urinary tract infections, a doctor will usually clear up any infection with antibiotics before treating the BPH itself. Although the need for treatment is not usually urgent, doctors generally advise going ahead with treatment once the problems become bothersome or present a health risk.

Drug Treatment

Finasteride, and dutasteride, inhibit production of the hormone DHT, which is involved with prostate enlargement. The use of either of these drugs can either prevent progression of growth of the prostate or actually shrink the prostate in some men.

Also Terazosin, doxazosin, tamsulosin, and alfuzosin are currently used for the treatment of BPH. All four drugs act by relaxing the smooth muscle of the prostate and bladder neck to improve urine flow and to reduce bladder outlet obstruction. The four drugs belong to the class known as alpha blockers. Terazosin and doxazosin were developed first to treat high blood pressure. Tamsulosin and alfuzosin were developed specifically to treat BPH.

Transurethral microwave procedures

In the procedure called transurethral microwave thermotherapy (TUMT), the device sends computer-regulated microwaves through a catheter to heat selected portions of the prostate to at least 111 degrees Fahrenheit. A cooling system protects the urinary tract during the procedure. The procedure takes about 1 hour and can be performed on an outpatient basis without general anesthesia. TUMT has not been reported to lead to erectile dysfunction or incontinence.

Transurethral needle ablation

The TUNA system delivers low-level radiofrequency energy through twin needles to burn away a well-defined region of the enlarged prostate. Shields protect the urethra from heat damage. The TUNA system improves urine flow and relieves symptoms with

fewer side effects when compared with transurethral resection of the prostate (TURP). No incontinence or impotence has been observed.

Water-induced thermotherapy

This therapy uses heated water to destroy excess tissue in the prostate. A catheter containing multiple shafts is positioned in the urethra so that a treatment balloon rests in the middle of the prostate. A computer controls the temperature of the water, which flows into the balloon and heats the surrounding prostate tissue. The system focuses the heat in a precise region of the prostate. Surrounding tissues in the urethra and bladder are protected. Destroyed tissue either escapes with urine through the urethra or is reabsorbed by the body.

High-intensity focused ultrasound

The use of ultrasound waves to destroy prostate tissue is still undergoing clinical trials in the United States. The FDA has not yet approved high-intensity focused ultrasound.

Surgical Treatment

Most doctors recommend removal of the enlarged part of the prostate as the best long-term solution for patients with BPH. With surgery for BPH, only the enlarged tissue that is pressing against the urethra is removed; the rest of the inside tissue and the outside capsule are left intact. Surgery usually relieves the obstruction and incomplete emptying caused by BPH. The following section describes the types of surgery that are used.

Transurethral surgery

In this type of surgery, no external incision is needed. After giving anesthesia, the surgeon reaches the prostate by inserting an instrument through the urethra.

A procedure called transurethral resection of the prostate (TURP) is used for 90 percent of all prostate surgeries done for BPH. With TURP, an instrument called a resectoscope

is inserted through the penis. The resectoscope, which is about 12 inches long and 1/2 inch in diameter, contains a light, valves for controlling irrigating fluid, and an electrical loop that cuts tissue and seals blood vessels.

Open surgery

In the few cases when a transurethral procedure cannot be used, open surgery, which requires an external incision, may be used. The location of the enlargement within the gland and the patient's general health help the surgeon decide which of the three open procedures to use. With the open procedures, anesthesia is given and an incision is made. Once the surgeon reaches the prostate capsule, he or she scoops out the enlarged tissue from inside the gland.

Laser surgery

The doctor passes the laser fiber through the urethra into the prostate using a cystoscope and then delivers several bursts of energy lasting 30 to 60 seconds. The laser energy destroys prostate tissue and causes shrinkage. As with TURP, laser surgery requires anesthesia and a hospital stay. One advantage of laser surgery over TURP is that laser surgery causes little blood loss. Laser surgery also allows for a quicker recovery time. But laser surgery may not be effective on larger prostates.

Photoselective vaporization of the prostate

PVP uses a high-energy laser to destroy prostate tissue and seal the treated area.

Interstitial laser coagulation.

Unlike other laser procedures, interstitial laser coagulation places the tip of the fiberoptic probe directly into the prostate tissue to destroy it.

Sexual Function After Surgery

Complete recovery of sexual function may take up to 1 year, lagging behind a person's general recovery. The exact length of time depends on how long after symptoms appeared that BPH surgery was done and on the type of surgery.

Need for the study and selection of methodology

Hyperplasia begins in the region of the veru-montanum, in the inner zone of the prostate, and extends to involve lateral lobes. This enlargement impinges upon the prostatic urethra, leading to the difficulty on urination with hesitency that is typical for this condition. Dysuria, dribbling, and nocturia are also frequent. The urinary tract obstruction leads to urinary retention and risk for infection. In severe, prolonged cases, hydroureter with hydronephrosis and renal failure can ensue. The main reason is cell multiplication during enlargement. With this logic this investigation was correlated in the DAL lymphoma cancer cell lines proliferation in animal models to ascertain the efficacy of the Velvanga parpam in the reduction of cell multiplication rate with respect to the prostatic hyperplasia.

MATERIALS AND METHODS

Animals Used

Swiss albino mice (20-25g) were used throughout the study. They were housed in standard microlon boxes and were given standard laboratory diet and water *ad libitum.*

Tumour cell lines

Dalton Ascitis Lymphoma (DAL) cells were obtained through the courtesy of Amala Cancer Research Centre, Thrissur, Kerala. DAL cells were maintained by weekly interaperitoneal (i.p) inoculation of 1 x 10^6 cells/mouse.

Effect of Velvanga Parpam on survival time

Animals were inoculated with 1x10^6 cells/mouse on day '0' and treatment with Velvanga Parpam started 24h after inoculation, at the doses of 1.5 and 3mg/kg/day orally. The control group was treated with same volume of 0.9% sodium chloride solution. All treatments were carried out for 9 days and observation was carried out for 45 days. The animals were subjected for the analysis of median survival time (MST) of each group (n=6) and changes in body weight. The antitumour efficacy of Velvanga Parpam was compared with that of 5 Fluorouracil (20mg/kg/day i.p. for 9 days). MST was noted with reference to control. Survival times of the treated group (T) were compared with those of the control groups (C) using the following calculation.

$$\text{Increase of life span} = \frac{T - C}{C} \times 100$$

Where T = number of days treated animals survived and C = number of days control animal survived.

Effect of Velvanga Parpam on haemotological parameters

In order to detect the influence of Velvanga Parpam on the haemotological status of DAL bearing mice, comparison was made amongst three groups (n=6) of mice on the 14^{th} day after inoculation. The three groups comprised (1) tumour bearing mice, (2) tumour bearing mice treated with Velvanga Parpam respectively (1.5 and 3mg/kg/day p.o. for first 9 days) and (3) control mice. Blood was drawn from each mouse in the conventional way and the white blood cell count, red blood cell count, haemoglobin, protein and packed cell volume were determined. The ascetic fluids were collected on 14^{th} day and smeared. The smear was stained with Giemsa stain for cytological studies.

Effect of Velvanga Parpam on solid tumour

Mice were divided into two groups (n=6). Tumour cells ($1x10^6$ cells/mice) were injected into the right hind limb of all the animals intramuscularly. Mice of group I were tumour control. Group II received Velvanga Parpam respectively (1.5 and 3mg/kg) orally for 5 alternate days. Tumour mass was measured from 11^{th} day of tumour induction and was repeated every 5^{th} day for a period of 30 days. The volume of tumour mass was calculated using the formula V-4/3 π r^2 where is the mean of r_1 and r_2 which are two independent radii of the tumour mass.

Statistical analysis

All the values were expressed as mean ± SEM. The data was statistically analyzed by one-way ANOVA followed by Dunnett's test. P values < 0.05 were considered significant.

RESULTS AND DISCUSSION

Antitumour studies

Mean Survival Time (MST)

Any potential anticancer drug is expected to increase the mean survival time and thus increasing life expectancy. Mice transplanted with DAL in our studies have MST of 22 days, which was increased to 26 and 34.32 days by Velvanga Parpam 1.5 and 3mg/kg respectively. These results are almost comparable to that of 5-FU, the standard drug for which the MST was 41.21 days.

Haematological Parameters

In malignancy there is always an alteration of various haematological parameters which increase in a few and decrease in others. There is a decrease in Hb, RBC and lymphocytes in malignancy accompanied by an increase in WBC especially Neutrophils, protein and PCV.

These changes are due to iron deficiency or due to haemolytic of myelopathic conditions induced by malignancy. Velvanga Parpam have very well reverted the above haematological parameters altered by the transplantable tumour of DAL. Velvanga Parpam may have direct tumoricidal effect and thereby maintain normal haematological profile.

Solid tumour volume

Estimation of solid tumour volume is a direct method of evaluation of anticancer activity. It is indeed a suitable method, which does not involve sacrificing the animal. In the study, the tumour mass was directly measured after implantation intramuscularly. The solid tumour volume was increased by 6.63±0.13 DAL bearing mice, treatment with Velvanga Parpam decreased significantly ($P<0.01$) the tumour volume to 4.15 ± 0.09 ml respectively on dose dependent manner at the end of 30 days.

The reliable criteria for evaluating an anticancer drug are prolongation of lifespan of the animal and decrease in WBC count of blood. Our results show an increase in life span accompanied by a reduction in WBC count in Velvanga Parpam treated mice. These results clearly demonstrate the antitumour effect of Velvanga Parpam against DAL.

The common problems encountered in cancer chemotherapy are myelosuppression and anaemia. Anaemia occurring in tumour bearing mice is mainly due to reduction in RBC or hemoglobin production, and this may occur either due to iron deficiency or due to haemolytic or other myelopathic conditions. Treatment with Velvanga Parpam brought back the hemoglobin content, RBC and WBC counts to near normal. This indicates that Velvanga Parpam have a protective effect on the haemopoietic system.

Further, analysis of haemotological parameters showed minimum toxic effect in mice treated with Velvanga Parpam. In DAL bearing mice, haematological parameters were reversed to normal by Velvanga Parpam administration (9 days).

Cytological studies of ascetic fluid on the 14th day in DAL bearing mice revealed that the tumour cells are large in size showed binucleation. In Velvanga Parpam 1.5 and 3mg/kg treated animals bearing DAL, the cells, showed plasmacytoid feature with varying degree of degeneration and cytoplasmic vacuolation and also showed active mitosis. All these cytological studies indicate the cytotoxic effect of Velvanga Parpam.

In DAL bearing mice, there was a regular and rapid increase in ascitic fluid volume. Ascitic fluid is the direct nutritional source for tumour growth, it meets the nutritional requirement of tumour cells. Velvanga Parpam treatment decreased the volume of solid tumour as well as ascites volume, viable cancer cell count and increased the life span. It may be concluded that Velvanga Parpam decrease the nutritional fluid volume and thereby arrest the tumour growth and increase the life span.

CONCLUSION

- In the present study the Velvanga Parpam was studied for its antitumour effect against transplantable tumour.
- The antitumor effect of the Velvanga Parpam is evident from the increase in lifespan, reduction in solid tumour volume and also the reversal of altered haematological parameter almost equal to normal.
- All these data confirms that the Velvanga Parpam can be used as a novel potential agent in the area of cancer chemotherapy.

REFERENCES

1. Anand V Naigaonkar and M.D Burande, 2004, A Manual of Medical Laboratory Technology, Nirali Prakasan. Pune. P 12-37.
2. Andríole G, Bruchovsky N, Chung LW, et al. Dihydrotestosterone and the prostate: the scientific rationale for 5alpha-reductase inhibitors in the treatment of benign prostatic hyperplasia. J Urol. 2004;172(4 Pt 1):1399-403.
3. Ansari, Mohammad Abduljalil; Begum, Dilruba; Islam, Fakhrul (2008). "Serum sex steroids, gonadotrophins and sex hormone-binding globulin in prostatic hyperplasia". Annals of Saudi Medicine 28 (3): 174–8.
4. Anti cancer activity of Ocimum sanctum, J of Pharma Biolo ., Vol 37 (4) , P 285-290.
5. Aua Practice Guidelines, Committee (2003). "AUA Guideline on Management of Benign Prostatic Hyperplasia (2003). Chapter 1: Diagnosis and Treatment Recommendations". The Journal of Urology 170 (2 Pt 1): 530–47.
6. Auffenberg GB, Helfand BT, McVary KT. Established medical therapy for benign prostatic hyperplasia. Urol Clin North Am. 2009;36:443-459.
7. Ayala AG, Ro JY. Prostatic intraepithelial neoplasia: recent advances. Arch Pathol Lab Med. 2007;131:1257-1266.
8. B Rajkapoor, B Jayakar and N Murugesh ,2004, Anti tumour activity of Indigofera aspalathoides on Ehrlich ascites carcinoma in mice.Indi.J.Pharmacol. Vol. 36, P 38-40.

9. Barry MJ, Fowler FJ, O'Leary MP, et al.(November 1992). "The American Urological Association symptom index for benign prostatic hyperplasia. The Measurement Committee of the American Urological Association". The Journal of Urology 148 (5): 1549–57; discussion 1564.
10. Bent, Stephen; Kane, Christopher; Shinohara, Katsuto; Neuhaus, John; Hudes, Esther S.; Goldberg, Harley; Avins, Andrew L. (2006). "Saw Palmetto for Benign Prostatic Hyperplasia". New England Journal of Medicine 354 (6): 557–66.
11. Black, L; Naslund, MJ; Gilbert Jr, TD; Davis, EA; Ollendorf, DA (2006). "An examination of treatment patterns and costs of care among patients with benign prostatic hyperplasia". The American journal of managed care 12 (4 Suppl): S99–S110.
12. Bostwick DG, Burke HB, Djakiew D, et al. Human prostate cancer risk factors. Cancer. 2004;101(10 Suppl):2371-2490.
13. Bostwick DG. Prospective origins of prostate carcinoma. Prostatic intraepithelial neoplasia and atypical adenomatous hyperplasia. Cancer. 1996;78:330-336.
14. Bostwick, D. G. (2002). "The Pathology of Benign Prostatic Hyperplasia". In Kirby, Peter; McConnell, John D.; Fitzpatrick, John M. et al.. Textbook of Benign Prostatic Hyperplasia. London: Isis Medical Media. ISBN 978-1-901865-55-4.
15. Boyle, P; Robertson, C; Lowe, F; Roehrborn, C (2000). "Meta-analysis of clinical trials of Permixon in the treatment of symptomatic benign prostatic hyperplasia". Urology 55 (4): 533–9.

16. Bushman W. Etiology, epidemiology, and natural history of benign prostatic hyperplasia. Urol Clin North Am. 2009;36:403-415.

17. Christensen, Tyler L.; Andriole, Gerald L. (February 2009). "Benign Prostatic Hyperplasia: Current Treatment Strategies". Consultant 49 (2): 115–22.

18. Dedhia, R; McVary, K (2008). "Phytotherapy for Lower Urinary Tract Symptoms Secondary to Benign Prostatic Hyperplasia". The Journal of Urology 179 (6): 2119-25.

19. Demura T, Shinohara N, Tanaka M, et al. The proportion of free to total prostate specific antigen: a method of detecting prostate carcinoma. Cancer. 1996;77:1137-1143.

20. Djavan, Bob; Marberger, Michael (1999). "A Meta-Analysis on the Efficacy and Tolerability of α_1-Adrenoceptor Antagonists in Patients with Lower Urinary Tract Symptoms Suggestive of Benign Prostatic Obstruction". European Urology 36 (1): 1–13.

21. Epstein JI. An update of the Gleason grading system. J Urol. 2010;183:433-40.

22. Feldman, Brian J.; Feldman, David (2001). "The development of androgen-independent prostate cancer". Nature Reviews Cancer 1 (1): 34–45.

23. Ganesh Chandra Jageti and Shival Kamalaksha Rao, 2006, evaluation of the antineoplastic activity of Guduchi(Tinospora cordifolia) in Ehrlich ascites carcinoma in bearing mice. J. Biol.Pharm.Bull. Vol. 29(3), P 460-466.

24. Gat, Y; Gornish, M; Heiblum, M; Joshua, S (2008). "Reversal of benign prostate hyperplasia by selective occlusion of impaired venous drainage in the male reproductive system: novel mechanism, new treatment". Andrologia40 (5): 273–281.
25. Gat, Yigal; Gornish, Michael (2009)."Reversal of Benign Prostate Hyperplasia by Super-selective Intraprostatic Androgen Deprivation Therapy". European Urology Review(Touch Briefings): 10–4.
26. Gilling, Peter J.; Aho, Tevita F.; Frampton, Christopher M.; King, Colleen J.; Fraundorfer, Mark R. (2008). "Holmium Laser Enucleation of the Prostate: Results at 6 Years". European Urology 53 (4): 744–9.
27. Gleason DF. Histologic grading of prostate cancer: a perspective. Hum Pathol. 1992;23:273-279.
28. Gormley, Glenn J.; Stoner, Elizabeth; Bruskewitz, Reginald C.; Imperato-Mcginley, Julianne; Walsh, Patrick C.; McConnell, John D.; Andriole, Gerald L.; Geller, Jack et al. (1992). "The Effect of Finasteride in Men with Benign Prostatic Hyperplasia". New England Journal of Medicine 327 (17): 1185–91.
29. Helpap BG, Bostwick DG, Montironi R. The significance of atypical adenomatous hyperplasia and prostatic intraepithelial neoplasia for the development of prostate carcinoma. An update. Virchows Arch. 1995;426:425-434.
30. Ho, C. K M; Nanda, J.; Chapman, K. E; Habib, F. K (2008). "Oestrogen and benign prostatic hyperplasia: effects on stromal cell proliferation and local formation from androgen". Journal of Endocrinology 197 (3): 483–91.

31. Homma Y, Kawabe K, Tsukamoto T, et al. Estimate criteria for diagnosis and severity in benign prostatic hyperplasia. Int J Urol. 1996;3:261-266.
32. Hutchison, A; Farmer, R; Verhamme, K; Berges, R; Navarrete, R (2007). "The Efficacy of Drugs for the Treatment of LUTS/BPH, A Study in 6 European Countries". European Urology 51 (1): 207–15 discussion 215–6.
33. Jung K, Lein M, Butz H, Stephan C, Loening SA, Keller T. New insights into the diagnostic accuracy of complexed and total prostate specific antigen using discordance analysis characteristics. J Urol. 2006;175:1275-1280.
34. K. Karthikeyan , P. Gunasekaran , N. Ramamurthy and S. Govindaswamy , 1999,
35. Kaplan, S. A.; Roehrborn, C. G.; Rovner, E. S.; Carlsson, M.; Bavendam, T.; Guan, Z. (2006). "Tolterodine and Tamsulosin for Treatment of Men With Lower Urinary Tract Symptoms and Overactive Bladder: A Randomized Controlled Trial". JAMA: the Journal of the American Medical Association 296(19): 2319–28.
36. Kaplan, S; McConnell, J; Roehrborn, C; Meehan, A; Lee, M; Noble, W; Kusek, J; Nybergjr, L et al. (2006). "Combination Therapy With Doxazosin and Finasteride for Benign Prostatic Hyperplasia in Patients With Lower Urinary Tract Symptoms and a Baseline Total Prostate Volume of 25 Ml or Greater".The Journal of Urology 175 (1): 217–20; discussion 220–1.
37. Lagiou, Pagona; Mantzoros, Christos S.; Tzonou, Anastasia; Signorello, Lisa B.; Lipworth, Loren; Trichopoubs, Dimitrios (1997). "Serum Steroids in Relation to Benign Prostatic Hyperplasia". Oncoogy 54 (6): 497–501.

38. Lieber, M M (1998). "Pharmacologic therapy for prostatism". Mayo Clinic Proceedings 73 (6): 590–6.
39. Lipski BA, Garcia RL, Brawer MK. Prostatic intraepithelial neoplasia: significance and management. Semin Urol Oncol. 1996;14:149-155.
40. MacDonald, Roderick; Wilt, Timothy J. (2005). "Alfuzosin for treatment of lower urinary tract symptoms compatible with benign prostatic hyperplasia: A systematic review of efficacy and adverse effects". Urology 66 (4): 780–8.
41. MacDonald, Roderick; Wilt, Timothy J.; Howe, R. William (2004). "Doxazosin for treating lower urinary tract symptoms compatible with benign prostatic obstruction: a systematic review of efficacy and adverse effects". BJU International 94 (9): 1263–70.
42. McVary, Kevin T.; Monnig, William; Camps Jr., Joseph L.; Young, Jay M.; Tseng, Li-Jung; Van Den Ende, Gene (2007). "Sildenafil Citrate Improves Erectile Function and Urinary Symptoms in Men With Erectile Dysfunction and Lower Urinary Tract Symptoms Associated With Benign Prostatic Hyperplasia: A Randomized, Double-Blind Trial". The Journal of Urology 177(3): 1071–7.
43. Mycek J , Richard A , Harvey A , Pamela , Champe C ,2000, In: Anticancer drugs – Pharmacology , Lippincot- Raven Publication , Philadelphia , NY , P 373-378.
44. Niu, YJ; Ma, TX; Zhang, J; Xu, Y; Han, RF; Sun, G (2003). "Androgen and prostatic stroma". Asian journal of andrology 5 (1): 19–26.
45. Page, S. T.; Lin, D. W.; Mostaghel, E. A.; Hess, D. L.; True, L. D.; Amory, J. K.; Nelson, P. S.; Matsumoto, A. M. et al. (2006). "Persistent Intraprostatic Androgen

Concentrations after Medical Castration in Healthy Men". Journal of Clinical Endocrinology & Metabolism 91(10): 3850–6.

46. Pearson JD, Luderer, Metter EJ, et al. Longitudinal analysis of serial measurements of free and total PSA among men with and without prostatic cancer. Urology. 1996;48(6A Suppl):4-9.
47. Potts JM. The four categories of prostatitis: a practical approach to treatment. Cleve Clin J Med. 2001;68:389-90, 392-3, 397.
48. Raminik sood, 1999, Medical Laboratory Technology. 5th edition ,P 404-414.
49. Roberts, Rosebud O.; Jacobson, Debra J.; Rhodes, Thomas; Klee, George G.; Leiber, Michael M.; Jacobsen, Steven J. (2004). "Serum sex hormones and measures of benign prostatic hyperplasia". The Prostate 61(2): 124–31.
50. Roehrborn, C; Boyle, P; Nickel, JC; Hoefner, K; Andriole, G; ARIA3001 ARIA3002 and ARIA3003 Study Investigators (2002). "Efficacy and safety of a dual inhibitor of 5-alpha-reductase types 1 and 2 (dutasteride) in men with benign prostatic hyperplasia". Urology60 (3): 434–41.
51. Roehrborn, C; Bruskewitz, R; Nickel, J; McConnell, J; Saltzman, B; Gittelman, M; Malek, G; Gottesman, J et al. (2004). "Sustained Decrease in Incidence of Acute Urinary Retention and Surgery With Finasteride for 6 Years in Men With Benign Prostatic Hyperplasia". The Journal of Urology 171 (3): 1194–8.
52. Roehrborn, CG (2008). "Current Medical Therapies for Men With Lower Urinary Tract Symptoms and Benign Prostatic Hyperplasia: Achievements and Limitations". Reviews in urology10 (1): 14–25.

53. Roehrborn, Claus G (2001). "Efficacy and safety of once-daily alfuzosin in the treatment of lower urinary tract symptoms and clinical benign prostatic hyperplasia: a randomized, placebo-controlled trial". Urology 58 (6): 953–9.
54. Roehrborn, Claus G.; Nuckolls, James G.; Wei, John T.; Steers, William; BPH Registry and Patient Survey Steering Committee (2007). "The Benign Prostatic Hyperplasia Registry and Patient Survey: study design, methods and patient baseline characteristics".BJU International 100 (4): 813–9.
55. Rubenstein, Jonathan; McVary, Kevin T. (6 February 2008). "Transurethral Microwave Thermotherapy of the Prostate (TUMT)". eMedicine.
56. V Ramnath, Girija Kuttan and Ramadasan Kuttan, 2002, Anti tumour effect of Abrin on transplanted tumours in mice.Indian J Physiol.Pharmacol, Vol 46(1), P 69-77.
57. Verhamme, K; Dieleman, JP; Bleumink, GS; Van Der Lei, J; Sturkenboom, MC; Artibani, W; Begaud, B; Berges, R et al. (2002). "Incidence and Prevalence of Lower Urinary Tract Symptoms Suggestive of Benign Prostatic Hyperplasia in Primary Care—The Triumph Project". European Urology42 (4): 323–8.
58. Wasserman NF. Benign prostatic hyperplasia: a review and ultrasound classification. Radiol Clin North Am. 2006;44:689-710.
59. Wasserman, Neil F. (1 September 2006). "Benign Prostatic Hyperplasia: A Review and Ultrasound Classification". Radiologic Clinics of North America 44 (5): 689–710.

60. Wei Wang, Qing-Long Guo, Qi-Dong You, Kun Zhang, Yong Yang, Jun Yu, Wei Liu , Li Zhao, Hong-Yan Gu , Yang Hu , Zi Tan and Xiao-Tang Wang, 2006, The Anticancer activities of Wogonin In Murine sarcoma S180 both In Vitro and In Vivo. . J. Biol.Pharm.Bull. Vol.29(6), P 1132-1137.
61. Wilt, Timothy J; Ishani, Areef; Rutks, Indulis; MacDonald, Roderick (2007). "Phytotherapy for benign prostatic hyperplasia". Public Health Nutrition 3(4A): 459–72.
62. Wilt, Timothy; Ishani, Areef; MacDonald, Roderick; Stark, Gerold; Mulrow, Cynthia D; Lau, Joseph; Wilt, Timothy (1999). Wilt, Timothy J. ed. "Beta-sitosterols for benign prostatic hyperplasia". Cochrane Database of Systematic Reviews (2): CD001043.
63. Wilt, Timothy; Ishani, Areef; Wilt, Timothy; Rutks, I; Stark, G (1998). Wilt, Timothy J. ed. "Pygeum africanum for benign prostatic hyperplasia". Cochrane Database of Systematic Reviews (1): CD001044.
64. Wilt, Timothy; MacDonald, Roderick; Ishani, Areef; Rutks, Indy; Stark, Gerold; Wilt, Timothy (1998). Wilt, Timothy. ed. "Cernilton for benign prostatic hyperplasia". Cochrane Database of Systematic Reviews (2): CD001042.
65. Wolf AM, Wender RC, Etzioni RB, et al. American cancer society guideline for the early detection of prostate cancer: update 2010. CA Cancer J Clin. 2010;60(2):70-98.

Table-1: Effect of Velvanga Parpam treatment on the survival of tumour bearing mice

Treatment	**MST (d)**	**Life Span (%)**
Tumour control (Saline 3 ml/kg. p.o)	22 ± 0.572	-
Velvanga Parpam (1.5mg/kg p.o)	26± 0.852**	15.38
Velvanga Parpam (3mg/kg p.o)	34.32 ± 0.654**	35.89
5-FU (20mg/kg i.p)	41.21 ± 0.442**	46.61

**P<0.01 Vs Tumour control; Data were analyzed by one way ANOVA followed by dunnet test. N = 6

Table-2: Effect of Velvanga Parpam on solid tumor volume

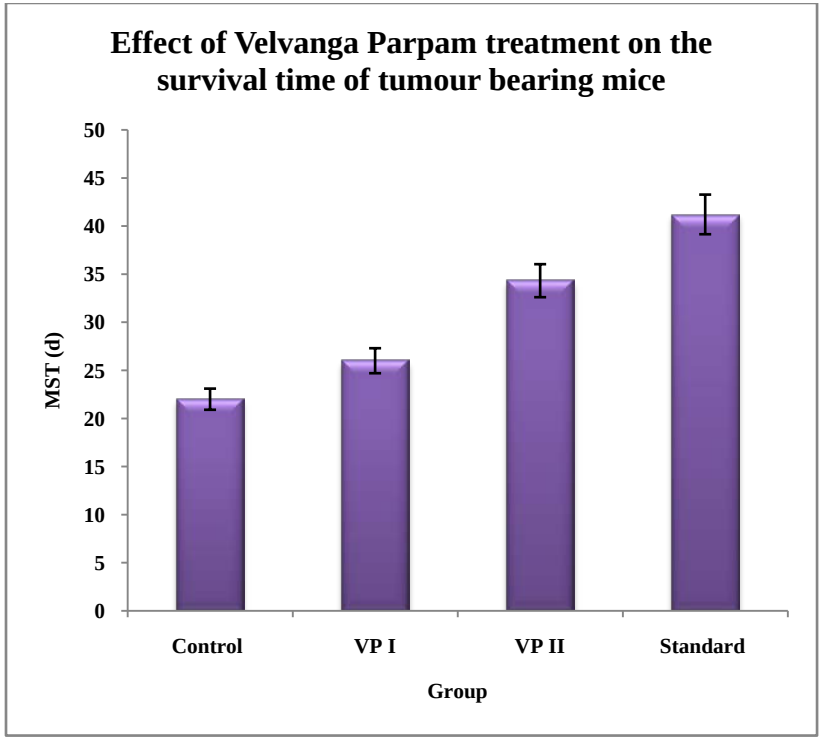
Treatment	**Dose (mg/kg)**	**Solid tumor volume (ml)**			
		15th Day	**20th day**	**25th day**	**30th day**
Tumor control	-	3.60 ± 0.131	4.10 ± 0.090	5.44 ± 0.210	6.63 ± 0.131
Velvanga Parpam	(1.5 p.o)	2.10 ± 0.052**	3.11 ± 0.062**	3.53 ± 0.091**	4.15±0.094**
Velvanga Parpam	(3 p.o)	1.79 ± 0.044**	2.42 ± 0.054**	3.14 ± 0.066**	3.42± 0.082**
5-FU	(20 i.p)	2.05 ± 0.035**	2.28 ± 0.035**	2.22 ± 0.052**	3.21 ± 0.060**

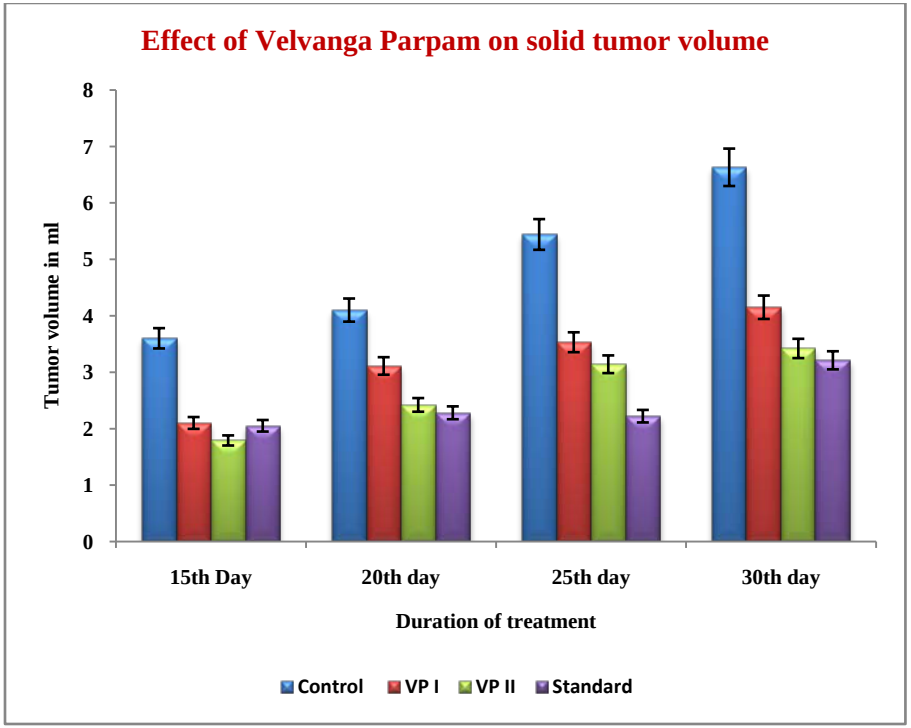
**P<0.01 Vs Tumour control; Data were analyzed by one way ANOVA followed by dunnet test. N = 6

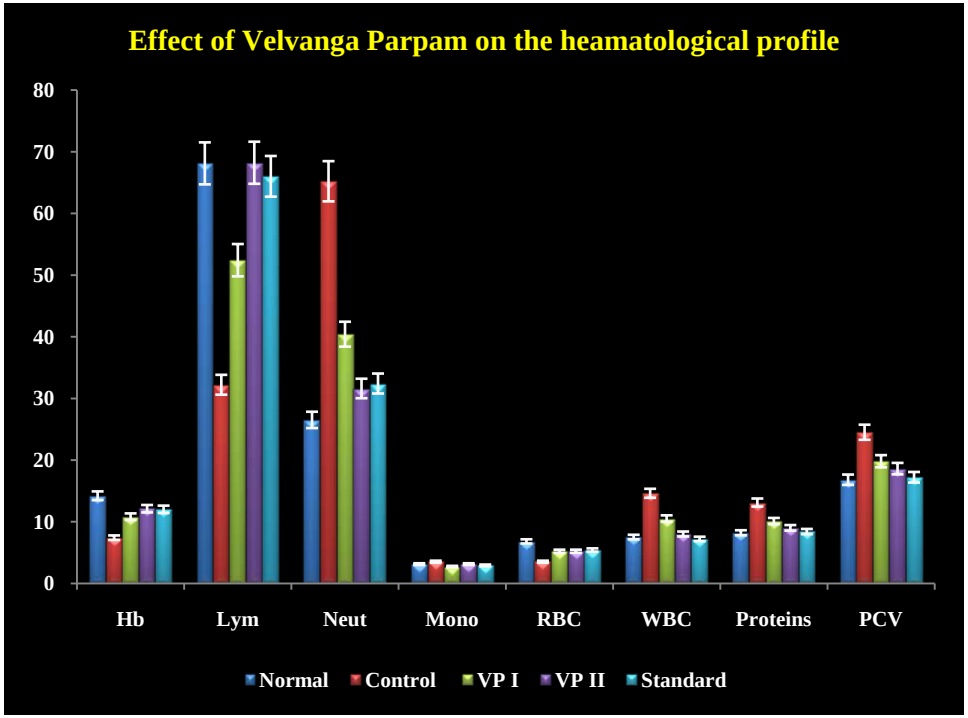
Table-3: Effect of Velvanga Parpam treatment on the heamatological profile of tumour bearing mice

Treatment	Hb (g%)	Differential count %			RBC (million/mm^3)	WBC (10^3cells/mm^3)	Proteins (g%)	PCV (mm)
		Lymphocyte	Neutrophil	Monocyte				
Normal saline (5 ml/kg)	14.2±0.4**	68.1±1.2**	26.5±1.4**	3.1±0.5	6.8±0.6**	7.5±0.5**	8.2±0.4**	16.8±0.5**
DAL control (1×10^6 cell)	7.4±0.5	32.2±0.2	65.2±1.6	3.5±0.4	3.5±0.2	14.6±1.0	13.1±1.2	24.5±0.3
DAL (1×10^6 cell) + VP (1.5mg/kg p.o)	10.8±0.4**	52.4±1.4**	40.4±1.5**	2.7±0.6	5.2±0.5*	10.5±0.5**	10.1±0.4**	19.8±0.5**
DAL (1×10^6 cell) + VP (3mg/kg p.o)	12.1±0.5**	68.2±2.3**	31.6±2.5**	3.1±0.4	5.2±0.3*	8.0±0.4**	9.0±0.2**	18.6±0.2**
DAL (1×10^6 cell) + 5FU (20 mg/kg i.p)	12.0±0.5**	66.0±2.0**	32.4±2.4**	2.9±0.4	5.4±0.2**	7.2±0.6**	8.4±0.3**	17.2±0.3**

**P<0.01 Vs Tumour control; Data were analyzed by one way ANOVA followed by dunnet test. N = 6


Effect of Velvanga Parpam treatment on the survival time of tumour bearing mice
MST (d)
50
45
40
35
30
25
20
15
10
5
0
Control
VP I
VP II
Standard
Group

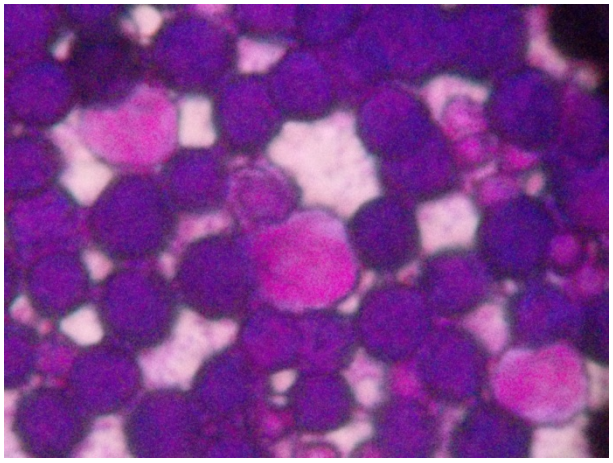

Effect of Velvanga Parpam on solid tumor volume
Tumor volume in ml
8
7
6
5
4
3
2
1
0
15th Day
20th day
25th day
30th day
Duration of treatment
Control
VP I
VP II
Standard

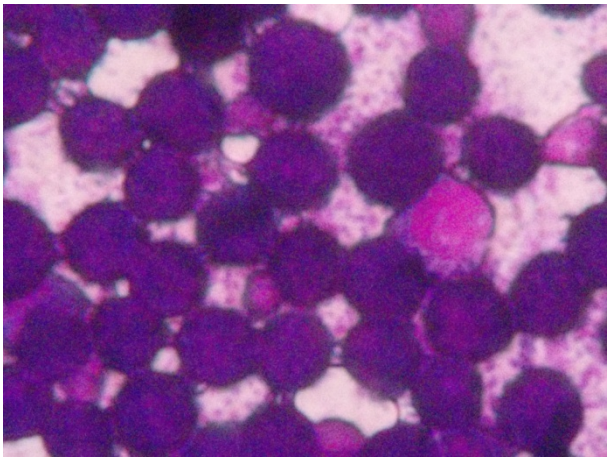

Effect of Velvanga Parpam on the heamatological profile
80
70
60
50
40
30
20
10
0
Hb
Lym
Neut
Mono
RBC
WBC
Proteins
PCV
Normal
Control
VP I
VP II
Standard

PHARMOCOLOGICAL STUDY OF VELVANGA PARPAM

HISTOPATHOLOGY

CONTROL:





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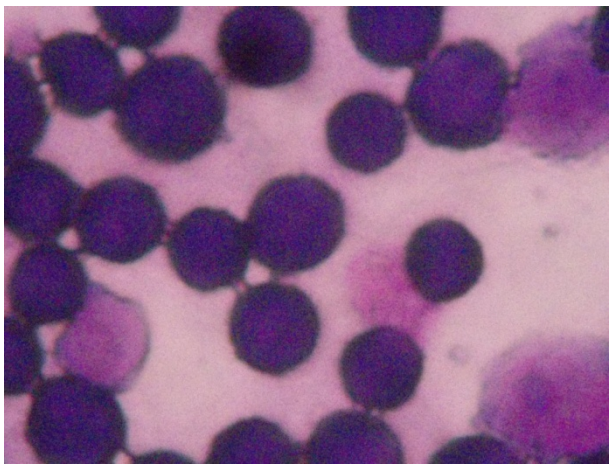

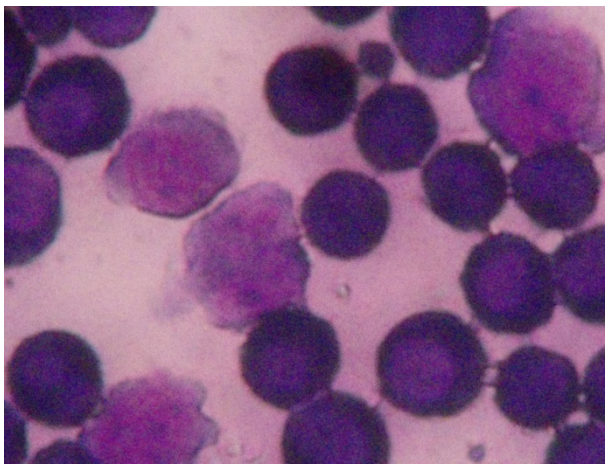

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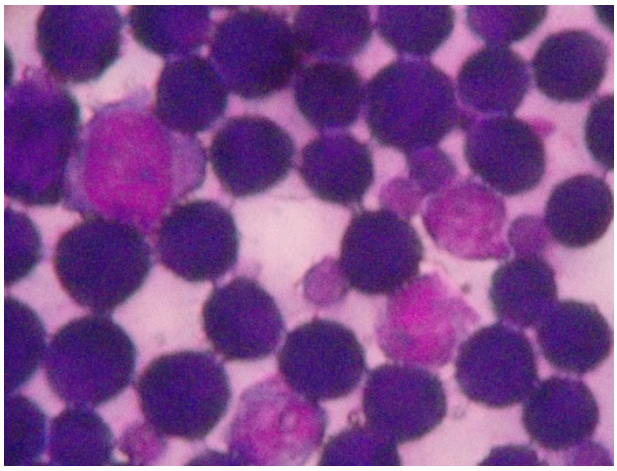
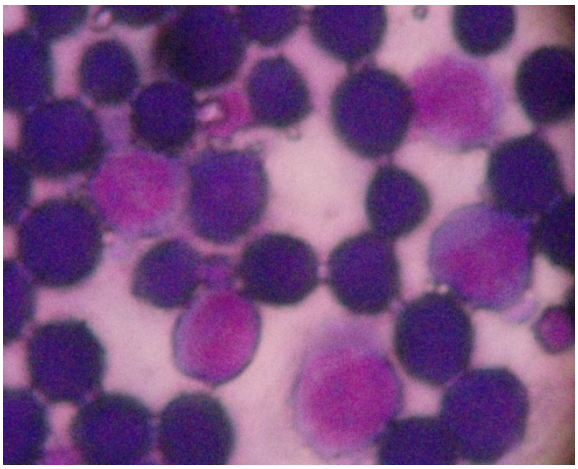
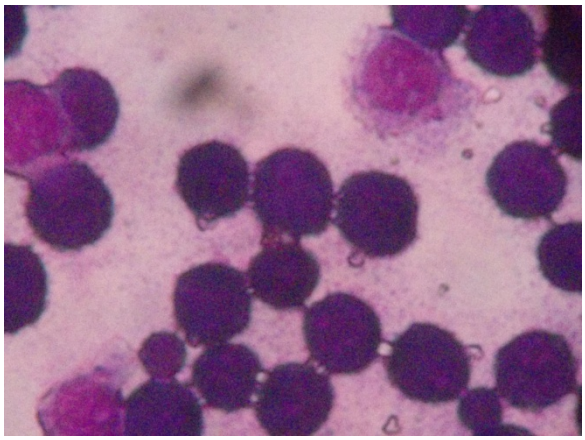
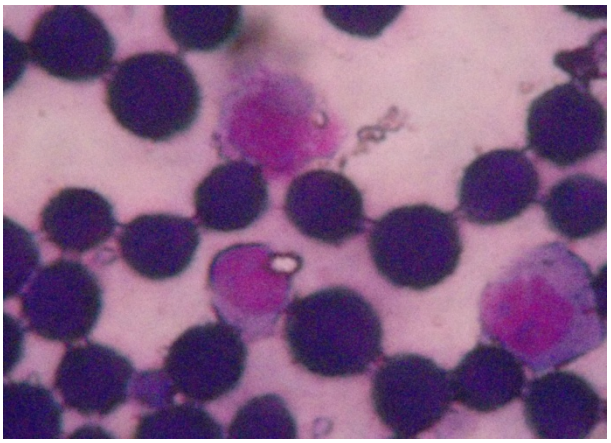




3 mg:





BIOSTATISTICAL ANALYSIS

ANNEXURE- IV

BIOSTATISTICAL ANALYSIS

Effect of Velvanga Parpam on IPSS in human subjects

S.No	IPSS	
	Before Treatment	After Treatment
1	7	5
2	19	12
3	32	28
4	18	10
5	17	7
6	17	12
7	15	7
8	11	6
9	19	13
10	14	7
11	13	6
12	7	4
13	28	12
14	24	16
15	16	9
16	16	8
17	13	8
18	10	5
19	27	24
20	14	7

Software: spss17 version

Variables: IPSS – before treatment, after treatment

Number of cases: 20

Test: Paired t test

Confidence Interval: 95%

Correlation coefficient (r): 0.893

Before and after treatment mean difference: -6.55±3.02

P Value (2 tailed): $p<0.01$.

Inference: The p value is significant ($p<0.01$). So the treatment was significantly reduces the symptoms score (IPSS).

Consent Form

ANNEXURE-V

CONSENT FORM

I certify that I have disclosed all the details about the study in the terms readily understood by the patient.

Date : **Signature**
Name :

Consent By The Patient

I have been informed to my satisfaction by the attending physician for the purpose of the clinical trial and the nature of the drug treatment and follow up including the lab investigation to be performed to monitor and safeguard my body functions.

I am aware of my right to opd out of the trial at any time during the course of the trial without having to give reasons for doing so.

I, exercising my free power of choice, hereby give my consent to be included as a subject in the clinical trial of **VELVANGA PARPAM** for the treatment of **UKKARA SOOLAI.**

Name : **Signature**

Date :

§¿¡Â¡ÇtÂtý ´ôÒ¾ø ÀÊÅõ

¾tÕ. _____ ¬¸tÂ ¿¡ý _____ÅÂÐ, (_____

_____

Åºtì Ìõ þ¼õ.) ±ý ÍÂ ¿t¨ É×¼ý ±Ø¾ì ¦¸¡Î ì Ìõ ´ôÒ¾ø ÀÊÅõ.

¿¡ý **ÒÃŠò¾ §¸¡Ç Åtì¸õ (¯ì¸¡Ã Ý¨Ä)** ±ý Ûõ §¿¡Â¡ø À¡¾ì¸ôÀð Î ¦ºý¨É,«ÃÍ ºtò¾ ÁÕòÐÅ ¸øæ¡Âtø (þ¼õ: «È t ï÷ « ñ ½¡ þó¾Â ÁÕòÐÅÁ¨É, «ÕõÀ¡ì¸õ, ¦ºý¨É-106.) ¿¼ò¾ôÀÎõ ºtò¾ ÁÕòÐÅ ¬Ã¡öîºt ãÄõ ºt¸tî¨º ¦ÀÈ ±ý ÍÂ ¿t¨É×¼ý ÓØºõÁ¾ò¨¾Ôõ ¦¾¡tÅtòÐì¦¸¡û¸t§Èý.

þó¾ ¬Ã¡öîºtÂtý §¿¡ì¸õ, ÁÕòÐÅõ ¦ºöÔõ Ó¨È, ¦¾¡¼÷¸ ñ ¸¡½tôÒ ÁüÚõ ±ý ¯¼ø ¿Äõ ÌÈtò¾ ÁÕòÐÅ À¡t§º¡¾¨É¸¨Çô ÀüÈtÂ Åt¡tÅ¡É ÅtÇì¸õ ±Éì Ì ÁÕòÐÅõ ¦ºöÔõ ÁÕòÐÅ÷ ãÄõ ¦¾Çt×ÀÎò¾ôÀðÎûÇÐ. þó¾ ¬Ã¡öîºtÂtø ÀíÌ¦¸¡ûÙõ ±ý ºõÁ¾ò¾üÌ Â¡Õ¨¼Â ¿t÷Àó¾Óõ ¸¡Ã½Átø¨Ä±ýÀ¨¾ ¦¾¡tÅtòÐì¦¸¡û¸t§Èý.

þôÀÊì Ì,

¦ÀÂ÷ :

Ó¸Å¡t :

¿¡û :

Case sheet proforma

ANNEXURE- VI

CASE SHEET
POST GRADUATE DEPARTMENT - BRANCH-I

(POTHU) MARUTHUVAM

GOVT. SIDDHA MEDICAL COLLEGE & ANNA HOSPITAL, CHENNAI-106.

CASE SHEET PROFORMA FOR "UKKARA SOOLAI"

WARD NO.	:	NATIONALITY	:
I.P. NO	:	RELIGION	:
BED NO	:	OCCUPATION	:
NAME	:	INCOME	:
AGE	:	D.O.A	:
SEX	:	D.OD	:
PERMANENT ADDRESS	:		
		DIAGNOSIS	:
TEMPORARY ADDRESS: Govt. Siddha Medical College & Anna Hospital, Chennai – 106.		MEDICAL OFFICER	:

COMPLAINTS AND DURATION :

HISTORY OF PRESENT ILLNESS:

HISTORY OF PAST ILLNESS :

PERSONAL HISTORY & HABITS :

1. Diet : Veg. ☐ Non veg. ☐
2. Marital status : single ☐ married ☐
3. Emotional stress : Yes ☐ No ☐
4. Addiction : Yes ☐ No ☐
   - If yes specify :_____
5. Bowel habit : Regular ☐ Constipation ☐
6. Sleep : Good ☐ Disturbed ☐ Insomnia ☐
7. Presence of anxiety : Yes ☐ No ☐

FAMILY HISTORY:

- Cardiovascular disease Yes ☐ No ☐
- Tuberculosis Yes ☐ No ☐
- Others Yes ☐ No ☐

If yes specify : .

GENERAL EXAMINATION:

1. Physical build : lean ☐ normal ☐ obese ☐
2. Height (cm) :
3. Weight(kg) : :
4. Pulse rate :
5. Heart rate :
6. Respiratory rate :
7. Blood pressure :
8. Pallor :
9. Cyanosis :
10. Jaundice :
11. Clubbing :
12. Pedal oedema :
13. JVP :

SYSTEMIC EXAMINATION:

- **CVS** : Normal ☐ Abnormal ☐
  - If abnormal , details_____
- **CNS** : Normal ☐ Abnormal ☐
  - If abnormal , details_____
- **Respiratory system** : Normal ☐ Abnormal ☐
  - If abnormal , details_____
- **Digestive system** : Normal ☐ Abnormal ☐
  - If abnormal , details_____
- **Urogenital system** : Normal ☐ Abnormal ☐
  - If abnormal , details_____

SIDDHA ASPECTS

Yaakai (udal nilai)

1. Vatham ☐
2. Pitham ☐
3. Kapham ☐
4. Kalappu ☐

Mukkunam

1. Sathuva gunam ☐
2. Raasatha gunam ☐
3. Thamo gunam ☐

PARUVA KAALAM (SEASONS)

1. Kaar Kaalam (Aavani-Puratasi) Aug-sept. ☐
2. Koothir Kaalam (Iypasi-Karthigai) Oct-Nov ☐
3. Munpani Kaalam (Maargazhi-Thai) Dec-Ja ☐
4. Elavenil Kaalam (Chithirai-Vaikasi) Apr-May ☐
5. Mudhuvenil Kaalam (Aani-Aadi) Jun-Jul ☐

NILAM (PLACES)

1.Kurinchi (Hills Areas) ☐
2.Mullai (Forest Areas) ☐
3.Marudham (Fertile Areas) ☐
4.Neithal (Sea Areas) ☐
5.Paalai (Desert Areas) ☐

IYAMPORIGAL/PULANGAL

1. Mei (Sensation) :
2. Vaai (Taste) :
3. Kann (Vision) :
4. Mooku(Smell) :
5. Sevi (Hearing) :

KANMENTHIRIYAM / KANMAVIDAYAM

1.Kai [Koduthal] :
2.Kaal [Nadathal] :
3.Vaai [Pesal] :
4.Eruvai [Malam Kazhithal] :
5.Karuvai [Aananthithal] :

MUMMALAM

1. Malam
2. Moothiram
3. Viyaravai

UYIR THATHUKKAL:

Vatham:

1. Pranan :
2. Abanan :
3. Viyanan :
4. Udhanan :
5. Samanan:
6. Naagan:
7. Koorman:
8. Kirukaran:
9. Devadathan:
10. Dhananjeyan:

PITHAM:

1. Anal Pitham:
2. Ranjaga Pitham:
3. Saadhaga Pitham:
4. Aalosaga Pitham :
1. Prasaga Pitham:

KAPHAM:

1. Avalambagam:
2. Kledagam:
3. Podhagam:
4. Tharpagam:
5. Santhigam:

UDAL THATHUKKAL:

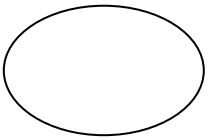
1. Saaram :
2. Senneer :
3. Oon :
4. Kozhuppu :
5. Enbu :
6. Moolai :
7. Sukkilam / Suronitham:

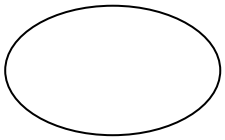
ENVAGAI THERVU:

1. Naa -
2. Niram -
3. Mozhi -
4. Vizhi -
5. Sparisam -
6. Malam
    a. Niram
    b. Nurai
    c. Erugal
    d. Elagal
7. Moothiram
    a. Neerkuri
        1. Niram
        2. Edai
        3. Manam
        4. Nurai
        5. Enjal
    b. Neikuri
8. Naadi

Neikuri examination:

Before treatment: **After treatment:**





SIGNS AND SYMPTOMS :

Date	Day	Wt	BP	Clinical Data	IPSS	Sign of M.O
	0					
	1st visit 15th Day					
	2nd visit 30th Day					
	3rd visit 45th Day					
	1st Follow up visit					
	2n Follow up visit					

LABORTORY INVESTIGATIONS:

Blood:

S.No	Parameters	Before treatment	After treatment
1.	**USG – KUB**		
2.	**Post void residual urine**		
3.	**PSA Test**		
4.	**TC**		

5.	**DC**		
6.	**Hb gms%**		
7.	**ESR 30 mints** **60 mints**		
8.	**Blood Sugar (F)** **(P)**		
9.	**S.Cholesterol**		
10.	**B.Urea**		
11.	**B.Creatinine**		

Urine:

s.no.	Parameters	Before treatment	After treatment
1.	**Albumin**		
2.	**Sugar**		
3.	**Deposits**		

TRAIL DRUG : VELVANGA PARPAM

Dose : 65 mg

Anubanam : Honey / butter

Duration of treatment: 45 days

Pathiam (Do’s and Don’ts)

Prognosis at the end of the treatment

Medical Officer Signature: **PROF. / H.O.D**

Bibliography

BIBILIOGRAPHY

Siddha Literature:

- ✓ Yugi vasithiya sinthamani
- ✓ Gunapadam mooligai vaguppu –Dr.K.S.Murugesa muthaliyar.
- ✓ Gunapadam thathu seeva vaguppu – Dr.R.Thiyagarajan L.I.M
- ✓ Therayar vagadam
- ✓ Agasthiyar gunavagadam
- ✓ Agasthiyar2000
- ✓ Pararasa sekaram
- ✓ Siddha maruthuvam
- ✓ Siddha maruthuvaanga surukkam
- ✓ Noi nadal noi mudhal nadal thirattu – part 1 & 2 – Dr.M.Shanmugavelu H.P.I.M
- ✓ Anubava vaithiya deiva ragasiyam
- ✓ Theran venba
- ✓ Siddha Aruvai Maruthuvam – Dr.K.S.Uthamarayan.H.P.I.M
- ✓ Agasthiyar kanma kaandam
- ✓ Bohar 700
- ✓ Agasthiyar rathna surukkam
- ✓ Heritage of siddha medicine
- ✓ History of siddha medicine
- ✓ Thirukkural
- ✓ Theran maga karisal
- ✓ T.v.saambasivam pillai tamil agarathy
- ✓ Tamil lexicon dictionary vol 1
- ✓ 20 th century tamil per agarathy

Modern books

- ✓ Rackel text book o family medicine
- ✓ Harrison text book of medicine
- ✓ Davidson text book of medicine
- ✓ Text book of medicine – kumar & clark
- ✓ Gayton's physiology
- ✓ Robins pathology
- ✓ Gray's Anatomy
- ✓ Cecil's text book of medicine

Articles:

- ✓ Serum prostate specific antigen as a bio marker for diseases progression in beningn prostatic hyperplasia
- ✓ Guideliness of beningn prostatic hyperplasia- j.de la rosette, g.alivizatos.
- ✓ Siddha system of medicine: A historical appraisal - K.H.Krishnamurthy and G.Chandra mouli. Indian journel of science.
- ✓ Indian journel of urology.

Net evidences:

- ✓ Pubmed.com
- ✓ Medscape.com.